THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—25TH YEAR. SYDNEY, SATURDAY, SEPTEMBER 10, 1938. No. 11.

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.

Diverticulosis and Diverticulitis, by C. BICKERTON BLACKBURN, M.D. .. 405
Diverticulosis and Diverticulitis from the Radiological Point of View, by H. R. SEAR .. 409
The Occurrence of Juxtaarticular Nodules in Australia, by FRANK J. FENNER .. 412
Some Aspects of Psittacosis and the Isolation of the Virus, by A. R. TREMAIN, B.Sc. .. 417
Some Observations on the Use of Protamine Zinc Insulin in the Management of Diabetes Mellitus, by EWEN DOWNIE, M.D., M.R.C.P. .. 421

REPORTS OF CASES—

A Case of Extreme Hypersensitivity to Insulin, by REG. S. ELLERY, M.D., and P. GUY REYNOLDS, M.B., B.S. .. 426
Case of Arachnoiditis of the Cerebello-Pontine Angle, Causing Trigeminal Neuralgia, by A. E. COATES .. 427

REVIEWS—

A History of Women in Medicine .. 427
Artificial Fever .. 428
Forensic Medicine .. 428

LEADING ARTICLES—

Medical Meetings .. 429

CURRENT COMMENT—

Reactions to Blood Transfusion .. 430
Epidemic Diarrhœa amongst the New-Born .. 431

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Radiology .. 432
Physical Therapy .. 433

BRITISH MEDICAL ASSOCIATION NEWS— Page.

Scientific .. 434

PROCEEDINGS OF THE ROYAL COMMISSION APPOINTED TO INQUIRE INTO MATTERS PERTAINING TO NATIONAL HEALTH INSURANCE 435

POST-GRADUATE WORK—

Melbourne Permanent Post-Graduate Committee .. 443

CORRESPONDENCE—

National Health Insurance .. 443
Anæsthesia in Pulmonary Tuberculosis .. 444
Schopenhauer and his Ill Health .. 444
A Warning .. 444
The Royal North Shore Hospital of Sydney .. 445
The Empire Rheumatism Council .. 445

MEDICAL SOCIETIES—

Repatriation Department Medical Officers' Association .. 445

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—

New South Wales .. 445

NOTICES .. 445

DIARY FOR THE MONTH .. 446

MEDICAL APPOINTMENTS .. 446

MEDICAL APPOINTMENTS VACANT, ETC. .. 446

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 446

EDITORIAL NOTICES .. 446

DIVERTICULOSIS AND DIVERTICULITIS.[1]

By C. BICKERTON BLACKBURN, M.D.,
Sydney.

WHILE diverticula may be found in all parts of the alimentary tract, I understand that at this meeting our field of discussion is limited to the part of it distal to the ileo-caecal valve, and, further, that we are concerned only with the acquired type of diverticulosis. This has its origin in the protrusion of the mucosa and submucosa, with at most a few stray muscular fibres, through the muscular coat of the bowel. The result is a more or less flask-shaped or bottle-shaped sac inclined to be constricted at the neck where the muscular fibres surround it.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 30, 1938.

It is important to the clarity of our discussion that we shall clearly distinguish between the mere presence of these sacs, a condition defined as diverticulosis, and the existence of inflammation in one or more of them, spoken of as diverticulitis. Diverticula varying in number from a single one to hundreds may be found anywhere in the area under review, including the *appendix vermiformis*; but they are most common in the sigmoid flexure of the colon and after that in the descending colon. Abell[1] says that 85% are present in these two situations, and that autopsy findings indicate that they occur in 5% of people over forty years of age. Rarely they are found at an earlier age, even in children; but only 16 of 1,574 patients referred to by Mayo[2] were under forty. Wakefield,[3] of the Mayo Clinic, stated that he thought that more than 10% of all patients over forty years of age whose colons were examined radiologically at the clinic had diverticulosis.

Ætiology.

Little definite is known about the ætiology. Men seem to be about twice as likely to get diverticula as women, and fat people slightly more likely than thin. Chronic constipation, abuse of purgatives, and spastic colon, have all been blamed; but all these are more common among women, who are least liable to be affected. With so little accurate knowledge, it will be perhaps best simply to put diverticulosis down as one of the many penalties of advancing years.

As to what proportion of those harbouring diverticula eventually develop diverticulitis it is, of course, impossible to say. Practically all people with clinical evidence of diverticulitis will be submitted to X ray examination, while those with simple diverticulosis will have this investigation carried out only when they have some other lesion in the gastro-intestinal tract. Brown and Marcley,[4] of the Mayo Foundation, reviewed 1,100 cases of diverticula of the colon seen at the clinic during a ten-year period, and found that in 376 of these diverticulitis was present, while in the remaining 724 there was simple diverticulosis only; but such figures have no statistical value, as these were all cases in which some alimentary disorder had led to the X ray investigation. Brown and Marcley followed up the history of 192 of these 724 cases of diverticulosis over a period of one to twenty years, and found that there was only one in which possible diverticulitis developed; they did not actually see it. All writers are agreed that it is very perplexing that while all diverticulitis must start from diverticula, diverticula are practically never seen to go on to diverticulitis. In my own practice I have not known of a case.

This immunity from inflammation is of particular interest, because X ray examinations indicate that diverticula empty slowly and retain barium long after the rest of the colon has been evacuated—occasionally for months. This suggests that during active contraction of the bowel, diverticula are more likely to be filled than emptied, and that emptying will be best accomplished while the bowel as a whole is comparatively relaxed.

It seems to be generally accepted that the explanation of the greater incidence of diverticulitis in the distal part of the colon lies in the fact that the fæces are more solid there. While there is probably much to be said for this thesis, it must not be forgotten that diverticula are much more common in this area, nor that fæces, however liquid, can become inspissated if retained long enough, as is well illustrated by the stercoliths found in the appendix.

It may be of interest to add that I have completely failed to find a single reference to the finding in one of these sacs of indigestible food residue, such as seeds *et cetera*.

Symptomatology.

Diverticulosis.

There are no symptoms due to diverticulosis, and it may be opportune to quote here Brown[4] who, after reviewing 1,100 cases, states: "I wish to urge caution not to be hasty in putting the blame for vague abdominal complaints on colonic diverticula that happen to be encountered during careful study." The possible presence of diverticula should no doubt be kept in mind by surgeons when operating on the colon in middle-aged people. Abell[1] refers to the risk of opening a diverticulum and contaminating the peritoneum when cutting through the mesoappendix. He reported one case of diverticulitis of the appendix with perforation, and states that diverticulosis was present in 1·89% of appendices removed surgically in the Presbyterian Hospital, New York, in one year.

Diverticulitis.

The type of symptoms met with in diverticulitis may vary within a wide range from mild to desperately severe. Mild and transitory attacks might occur without notice if pain is absent, and quite possibly many are overlooked. Persistent pain in the lower left quadrant of the abdomen in a patient over forty years of age should always raise suspicion of diverticulitis, especially if it is worse before a bowel action or after a meal, when the gastrocolic reflex comes into play. Constipation is common; but quite often there is an increased irritability of the bowels, with rather frequent small relaxed stools, each leaving the feeling that evacuation of the bowel is incomplete. Careful inspection may discover on occasions a little pus and a streak or two of blood on the motion; but this is not characteristic. There are usually definite malaise and a rise of temperature to 37·5° C. or 38·4° C. (99·5° F. or 101° F.) and a frank neutrophile leucocytosis.

Palpation in such mild cases may reveal at most a vague "doughy" thickening somewhere in the course of the iliac colon or, if the lesion is low in the sigmoid, there may be tenderness and a sense of resistance between the finger in the rectum and the hand pressing down from above.

Till a barium enema has been given such cases are apt to be regarded as mild ulcerative colitis. But the diarrhœa, when present, is not severe; there is little, if any, mucus, and blood, if present, rarely consists of more than a trace, while sigmoidoscopic examination fails to reveal the characteristic congested friable mucosa in the upper part of the rectum and lower part of the sigmoid. The X ray examination is usually decisive, and shows the lumen of the involved area irregularly encroached on by the swollen mucosa.

In the more commonly diagnosed cases, by the time they are first seen, the inflammation has already extended well beyond the affected diverticulum into the neighbouring bowel wall, setting up something akin to a cellulitis that has resulted in an elongated sausage-shaped tumour. This is most commonly in the left iliac fossa, feels very solid, and usually appears fixed to the ilium; but when lower down may be felt *per rectum* as a definite mass between the two hands.

The chief complaints are general malaise, pain in the left lower part of the abdomen, occasionally slight chilly feelings, indigestion and wind in the bowel that is difficult to expel. Constipation and a sense that the bowel does not get properly emptied are usual; but sometimes frequent feebly successful efforts to relieve this lead to the idea that there is diarrhœa, while the straining may set up some proctitis, with discharge of blood and mucus, and so arouse suspicion of malignancy. Frequency of micturition, with some dysuria, is not uncommon, and at times leads to an early view that the lesion is in the urinary tract. It is rather curious that this symptom may appear in cases in which recovery is complete, and there is no evidence that the bladder has been directly involved.

Further investigation usually reveals a moderately raised temperature and a leucocytosis, while X ray examination after a barium enema shows great narrowing of the lumen of the affected part of the bowel, with often a collection of gas in the section above it. Sigmoidoscopic examination in my experience has been of value mainly in revealing other lesions; but some writers have actually seen diverticula, while Bockus[5] lays stress on the presence of spasm and œdema blocking the view of a low diverticulitis and the absence of this feature in carcinoma at the same level. Occasionally sudden complete obstruction is the first indication that anything is wrong; this is probably due to œdema of the mucosa rapidly completing a closure that has been gradually taking place from a painless pericolitis. In other rare instances an individual is abruptly plunged from health to dire peril through perforation of an unsuspected diverticular abscess, leading to local or general peritonitis.

Complications.

Complications that may occur in a case under observation include everything that can happen when ulceration starts in the wall of a hollow viscus containing infective material. These possibilities include: local and general peritonitis; widespread cellulitis, with or without single or multiple abscesses, which may reach the surface of the body or discharge into the colon or other portion of the bowel or the bladder; abscess of the liver, and general pyæmia.

I have included hæmorrhage other than slight as a complication. I saw one very severe case, and Brown and Marcley[4] record one in which the bleeding was found after resection of the bowel to have come from an angioma. Friedenwald and Feldman,[6] however, speaking of symptoms, say: "Blood is not very unusual, sometimes quite a smart bleeding."

Carcinoma was at one time regarded as a complication; but this view is now generally discarded and most writers support the view of Brown and Marcley[4] that "the relationship between diverticulitis and carcinoma is incidental rather than actual".

Prognosis.

The prognosis in diverticulosis is difficult to assess, since, as has already been pointed out, it has so rarely been known to be followed by diverticulitis. From the psychic point of view the outlook largely depends upon the significance attached by the family attendant to the discovery of this anatomical abnormality. I have seen several patients bearing X ray reports couched in such terms as, "No organic disease revealed in the alimentary tract, some diverticula present in the sigmoid colon", who were happily unhappy in the recent acquisition of diverticulitis. It is perhaps unfortunate that the suggestion made with singular prescience by Sir Edmund Spriggs[7] as long ago as 1929 to substitute for these cumbrous terms such variants of the English word sac as saccosis and saccitis, was not adopted; for the saccitis of one of its members could never be such an object of family pride as diverticulitis.

In diverticulitis proper the prognosis as regards life depends mainly upon whether complications arise requiring surgical intervention. In the series of cases referred to by Brown and Marcley,[4] and that of Friedenwald,[8] surgical intervention was required in about 16%. Of those patients treated by medical means, about 60% recover completely, and follow-up shows that in such cases relapse very rarely takes place. In my own limited series I know of no instance. Of the 40% that do not completely recover, statistics seem to indicate that about three-quarters get along in comparative comfort if they are reasonably careful as to their mode of life. It is interesting to note that among the cases recorded as cured by surgical measures any further operative treatment has very rarely been necessary.

Diagnosis.

Diverticulosis, being symptomless, will always be an accidental discovery, and the chief diagnostic difficulty will arise from the tendency to saddle the diverticula with responsibility for the condition for which the X ray examination was carried out, as, for example, constipation, morning diarrhœa, spastic colon *et cetera*.

When I was preparing these notes a patient consulted me and told me how she was daily seized with diverticulitis about 6.30 a.m., and how it accompanied her on several hurried visits till about 11 a.m., and then left her till the following morning.

In the case of diverticulitis, on the other hand, the X ray examination is usually carried out after a clinical diagnosis of the lesion has been made.

Pitfalls in diagnosis are a left-sided appendix with transposition of the viscera, and early bladder symptoms in middle-aged women with latent but unrelated chronic cystitis. In one case seen by me prostatitis had been confidently diagnosed. If there is no frank tumour and some blood-stained pus or mucus in the stools, ulcerative colitis may be suggested. When there is a tumour malignant disease is naturally suspected at this period of life, and the differentiation may be extremely difficult. Even

after sigmoidoscopic and Röntgenographic investigations have supplemented careful clinical examination and an obstruction has been relieved by an artificial anus, a diagnosis of carcinoma may still be wrong, as has been shown by the subsequent history of two cases that have come under my personal observation. From the clinical point of view, tenderness and early fixation of a swelling that appears elongated rather than globular, suggest diverticulitis, as also do fever—especially with chills—and leucocytosis, while loss of weight and the presence of blood and mucus in the stools favour a neoplasm. However, none of these features are pathognomonic, and those usual in one lesion may at times be found in the other. A wise clinician will approach each case with an open mind, and not put the whole responsibility for the diagnosis on the shoulders of the radiologist, even if he is willing to carry it. Less difficulty should be found in excluding compression of the colon by tumours arising in or obtruding into the pelvis, and such rarities as invasions of the bowel wall by tuberculosis, syphilis or actinomycosis.

Treatment.

Diverticulosis is not amenable to treatment, unless we accept the advice given in the 1926 edition of "Osler's Modern Medicine"[9] in regard to intestinal diverticula, which summarily dismisses treatment with the remark: "The treatment of all forms of diverticula is purely surgical." Diverticulitis seems to supervene so infrequently that the discovery of diverticula is probably a misfortune rather than a boon. For my part, if the patient knows about them I endeavour to impress upon him that the small pockets seen in his bowel are very unlikely ever to affect his health, but that it would be wise to avoid taking aperients and perhaps large-seeded berries, and to endeavour to correct any tendency to constipation by regular habits and, if necessary, by the use of agar or paraffin.

Authorities seem to be generally agreed that medical measures take first place in the modern treatment of diverticulitis. In the treatment of a definite attack, complete rest plays a very important part, and should be continued till some time after all symptoms have completely subsided. This may in some cases entail remaining in bed for many weeks, after which activity should be only very gradually resumed.

If there is a tumour causing partial obstruction, every effort must be made to avoid the necessity of a colostomy, since usually even after the operation the lumen of the bowel is restored as the œdema and inflammation subside.

In order to minimize peristaltic movements nothing at all should be given by mouth for the first two or three days, fluids being supplied by the subcutaneous or intravenous route. After this, fluids, glucose and easily assimilable foods should be sparingly given, and the quantity only very gradually increased as improvement takes place. Evacuation of fæces and gas from above the obstruction can usually be secured by the injection of small quantities of warm saline solution, supplemented, if need be, by warm olive oil injections given slowly in the knee-elbow position. I personally believe that the application of heat over the mass is useful, not only in lessening discomfort, but also by helping to relax spasm. Dry heat by means of electric pads is excellent; but if these are not available the frequent application of light hot foments seems to be very successful. Rest and mental quietude are important, and can usually be best secured by small repeated doses of phenobarbitone; but morphine will at times be needed.

In my own experience, if obstruction has not been complete when the patient has been put to bed, resolution has always taken place without operation, and where complete obstruction has been present it had existed before treatment was started.

After recovery the question arises as to what steps should be taken to prevent a recurrence. Fortunately, as has been already indicated, those writers who have recorded large series of cases are in agreement that those who completely recover do not seem especially liable to have further attacks. This possibly explains why success in preventing recurrences is confidently claimed by the use of such varied diets as: "a concentrated nutritious diet leaving little residue"; "mainly vegetarian, but excess cellulose must be avoided"; "coarse cereals and vegetables form an important part of the dietary". Again, success is claimed for the certainly ingenious, if unconvincing, treatment referred to in "Nelson's Living Medicine"[10] as being of "extreme value"—the administration of large doses of bismuth subcarbonate, one ounce a week, on the principle that "something must fill these pockets, and a non-toxic, unabsorbable, bland substance would seem to be preferable to putrefactive fæces".

The knowledge that there is such a wide choice in methods has fortified me in my belief that these people will probably manage to take an average normal diet as well as anyone else, and I shall continue to advise them to eat just what their previous experience has shown suits them, adding as a gesture that they had better abstain from nuts and berries.

Where, however, recovery is incomplete, it is quite another matter, and in these cases dieting is an individual question in which idiosyncrasy and coincident digestive disabilities will have to be taken into consideration.

In the after-care of all cases attention will have to be paid to the bowels, since it is obviously undesirable that the sigmoid should be allowed to become a repository for caked fæces. Probably the most important instruction we can give is a negative one: not to take purgatives. Where it is the habit to have a relaxed daily evacuation nothing is to be gained by interfering. But often it will be found that the normal rhythm has been completely disorganized by prolonged abuse of aperients. In many of these cases excellent results will be obtained by increasing the bulk of the stools by such materials as agar-

TABLE I.

Results of the Analysis of the Faeces before and after Administration of Olive Oil.

Patient.	Water Percentage.	"Total Fat" Percentage of Dried Faeces.	"Split Fat" (as Stearic Acid) Percentage of Dried Faeces.	Before or After Oil.	Condition of Faeces.
B	(a) 92·0	21·7	11·0	Before.	Very fluid.
	(b) 85·4	15·6	9·6	"	Soft, not well formed.
	(c) 74·8	23·1	14·1	After.	Normal consistency, well formed.
	(d) 73·3	24·5	14·6	"	Well formed, tending to hardness.
	(e) 76·0	26·2	16·4	"	Well formed, normal consistency.
C	(a) 92·2	19·5	9·5	Before.	Very fluid.
	(b) 88·2	20·8	7·0	"	Fluid.
	(c) 79·6	14·2	7·9	"	Rather soft, not well formed.
	(d) 77·1	14·9	7·8	After.	Very soft, but not fluid, unformed.
	(e) 90·2	12·5	4·4	"	Very fluid.
M	(a) 60·8	27·8	19·7	Before.	Well formed, normal consistency, tending to firmness.
	(b) 79·9	22·6	12·9	"	Rather soft, not well formed.
	(c) 83·1	22·8	14·5	"	Soft, not well formed.
	(d) 96·6	15·6	10·2	After.	Very fluid.
	(e) 71·2	27·4	18·4	"	Well formed, tending to firmness.
	(f) 70·7	26·4	17·2	"	Well formed, normal consistency.

agar or the proprietary preparation "Normacol". For those who find these too expensive, plain paraffin will usually act admirably, though occasionally this comes away separately and sometimes seems to encourage the formation of pellet-like stools that suggest the rôle of diverticular plugs.

Brown and Logan[11] extol the virtues of olive oil as a lubricant in these cases, and suggest that, although it is a foodstuff and so absorbable, it probably increases the fat in the stools. As quite recently I was told by a man who on lay advice had treated his indigestion by consuming a gallon of olive oil in three months, that though he had gained a good deal of weight he had observed no change in his motions, I thought it might be of interest to try to determine the fate of olive oil in a few normal people. Dr. E. B. Jones kindly arranged to have some investigations carried out in the biochemical department of the Prince Henry Post-Graduate Hospital, using as subjects some otherwise normal fracture patients. Mr. R. J. Bartholomew, B.Sc., who kindly carried out this rather tedious investigation on three patients, reports as follows:

The general procedure was to collect a specimen from each patient on each of six consecutive days, each patient taking one ounce of olive oil at 4 a.m. on the fourth, fifth and sixth days of the series. I have since found that unfortunately the patients may have had magnesium sulphate during the first three days of the experiment, so that no inference can be drawn from comparison of the macroscopic appearances of the specimens before and after the olive oil. It can be said, nevertheless, that the "after-oil" specimens showed no regular alteration from normal. The results of the analyses are tabulated below, and from these it will be seen that there is no regular characteristic difference between the "before" and "after" figures for each patient.

It would be useful to compare these results with some stools obtained under similar circumstances from patients taking paraffin; but the investigation involves a great expenditure of time. It may, however, be deduced that in these three people at least the olive oil was largely assimilated and that there was no indication that its ingestion produced stools that would be especially unlikely to form fæcal concretions in diverticuli. It is noteworthy that whether oil was taken or not the consistency of the stools was conditioned by the water in them rather than by the fat.

References.

(1) Abel Irvin: "The Diagnosis and Treatment of Diverticulitis and Diverticulosis", *Surgery, Gynecology and Obstetrics*, Volume LX, 1935.
(2) William J. Mayo: "Diverticulitis of the Sigmoid", *The British Medical Journal*, September 28, 1929, page 574.
(3) E. G. Wakefield: Personal communication.
(4) P. W. Brown and D. M. Marcley: "Prognosis of Diverticulitis and Diverticulosis of the Colon", *The Journal of the American Medical Association*, Volume CIX, October 23, 1937, page 1328.
(5) H. L. Bockus: *The Journal of the American Medical Association*, Volume CIX, October 23, 1937, page 1333.
(6) J. Friedenwald and M. Feldman: "Diverticulosis and Diverticulitis", *Review of Gastroenterology*, Volume II, June, 1935, page 97.
(7) E. I. Spriggs: "Diverticulitis", *The British Medical Journal*, Volume II, September 28, 1929, page 569.
(8) J. Friedenwald: *The Journal of the American Medical Association*, Volume CIX, October 23, 1937, page 1333.
(9) McCrae: "Osler's Modern Medicine", Volume III, page 630.
(10) "Nelson's Living Medicine."
(11) P. W. Brown and A. H. Logan: *Proceedings of Staff Meetings of the Mayo Clinic*, Volume II, Number 3, page 39.

DIVERTICULOSIS AND DIVERTICULITIS FROM THE RADIOLOGICAL POINT OF VIEW.[1]

By H. R. SEAR,

Sydney.

IT is my pleasure and duty to put before you tonight a brief survey of the subject "diverticulosis and diverticulitis" from the radiological aspect. I feel that but little is left to the radiologist after the dissertation given on the historical, pathological and clinical aspects. I could collect statistics; but this has already been done over a much greater series of cases than are available to me.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 30, 1938.

It has been held that impressions are often very erroneous, that statistics alone are reliable. In spite of this I propose to give the impressions that I have formed in the course of my practice as a radiologist.

In my early days of X ray work diverticula had not been described radiographically. It was not till the articles of Carman (*American Journal of Roentgenology*, Volume II, 1915), Case (*Archives of Roentgen Rays*, April, 1915) and the books of Carman and Miller, A. W. George and R. D. Leonard, that this clinical entity was recognized, I think, by most of us radiologists. I know that the earlier description by Abbé escaped my notice, nor did I hear anything of it from my colleagues. Since those days a great number of articles have appeared, with imposing statistics, but little has been added to the radiological side (*vide* Carman and Miller).

Diverticula show radiographically as projections from the opaque shadow of the contrast-filled bowel, more or less oval or rounded, sometimes sessile, sometimes pedunculated. Usually small in size, they may in rare cases be 2·5 centimetres (one inch) in diameter. There may be a single diverticulum, or they may range in number to several hundreds. The necks of the diverticula, their openings into the bowel, may be wide or almost stenosed. They may be only partly filled with the opaque substance, the latter flowing round an inspissated fæcal mass in the diverticulum, giving a crescentic shadow. They are commonest in the sigmoid colon, but may be seen anywhere along the large bowel. I have seen them associated with numerous diverticula in the small bowel, the condition being associated with many adhesions. They are frequently scattered as isolated irregularities along the whole colon.

The appendix has been described by Moynihan as a common site. There seems to be some contention as to the frequency of diverticula arising in the appendix, as seen at operation and in the *post mortem* room. Speaking as a radiologist I should say that it is rarely observed during X ray examination of the gastro-intestinal tract.

The diverticula may retain their contents, opaque meal or otherwise, for a long time. If packed with non-opaque material they may not admit any contrast substance or may admit only a little, giving a crescentic shadow. This non-filling and partial filling raised the question which raged for some time, of whether they are best demonstrated by opaque meal or opaque enema. As late as 1933 Barclay wrote:

> These fill with the opaque food far more readily than with the enema. Why this should be is not clear, but I have frequently failed to demonstrate diverticula with the enema, and conversely the enema has seldom revealed diverticula that had not been filled with the opaque food. Reexamination after an enema has been voided will, however, sometimes show diverticula that were not seen when the bowel was filled.

My view is that diverticula will be revealed by either examination, meal or enema, but that the enema is essential, for the following reasons: most of these people are submitted not only to prove or disprove the presence of diverticula, but also to exclude, as far as possible, carcinoma; and for the latter an enema is essential. Also one may see with a meal irregular haustration which may simulate diverticula, which are proved non-existent by the enema. As mentioned before, diverticula may be packed with material, such as tomato skins, that renders them difficult to demonstrate by either means, though I do not think that this is common. I have seen one, in the transverse colon, cast a faint shadow that simulated a gall-stone, in the days prior to the Graham's test.

In the early period we spoke of "diverticula" and "diverticulitis" and sometimes of "peri-diverticulitis". Later came the term "diverticulosis". Personally I have regretted the term "diverticulosis". Too often it is mistaken by the medical attendant for "diverticulitis". An excellent example of such confusion occurs in Barclay's book "The Digestive Tract", when he says: "Diverticulitis is a symptomless condition." Diverticulitis is anything but symptomless. One can readily realize how such confusion has arisen; for Spriggs and Marxer, in their article "Intestinal Diverticula" (*The British Medical Journal*, January 23, 1926), give the following classification:

(1) Prediverticular state (in colon) (2) Quiescent diverticulosis (3) Diverticulitis	diverticulosis

This was later modified by Spriggs (*The British Medical Journal*, September, 1929):

(1) The prediverticular state (2) The stage of formed diverticula	diverticulosis
(3) The stage of diverticulitis or inflamed diverticula	

In my reports I usually avoid the word diverticulosis, reporting usually scattered diverticula, numerous diverticula, isolated diverticula, as the case may be, in such-and-such a part of the colon. I feel that the radiologist may be unwittingly often the cause of much anxiety to the patient through the confusion of the terms "diverticulosis" and "diverticulitis". The percentage of middle-aged people who have diverticula without symptoms is so large that it is possibly advisable that they should not know of their presence. This, however, is out of my sphere, as it is left always to the physician or surgeon to acquaint the patient of the X ray findings.

The next question to be considered is the frequency of diverticula and diverticulitis, and with it also of carcinoma. Spriggs and Marxer (*The British Medical Journal*, January 23, 1926) state that diverticula were observed in 100 out of 1,000 X ray examinations of the colon, and that the average age of patients with diverticula was fifty-eight years. In the Mayo Clinic the figure was definitely smaller—5%. Robert Mailer (*The Lancet*, July 14, 1928) says that "one could say roughly that 13% or one person in eight over the age of 45, showed

diverticular formation in the large bowel". Mayo's figures ("Diverticulitis of the Sigmoid", *The British Medical Journal*, September 28, 1929) are very interesting:

> Of our 1,918 recorded cases of diverticulosis, active diverticulitis was present in 653 at the time the patients were examined.

This raises the point of what constitutes the radiographic features of active diverticulitis. Is the diagnosis to be based on radiographic evidence, spasticity and narrowing of the lumen of the colon, and flattening and deformity of the pockets by swelling of the mucosa of the bowel wall? Or do we have to deny this and leave the evidence of the activity of the process to the medical attendant, who bases it on symptoms, tenderness, mass formation *et cetera*? Speaking of diverticulitis Spriggs says:

> Strictly speaking, whenever any little pouch is the seat of inflammation, the patient is the subject of diverticulitis. But for clinical purposes we reserve the terms for the stage at which inflammation arising from a diverticulum has spread to a considerable area of the wall of the bowel, involving most, if not all, of its thickness.

If we base the diagnosis on the radiological evidence, I should say that in my experience the percentage of cases of diverticulitis compared with those of simple diverticula is much smaller than that quoted by Dr. Mayo, who found diverticulitis in 653 out of 1,918 cases, or approximately one in three. I find uncomplicated diverticula common, and obvious diverticulitis not nearly so common. I shall say more of this when dealing with carcinoma of the colon.

The next point I want to discuss is the prediverticular state. Spriggs and Marxer wrote (*The British Medical Journal*, January 23, 1926):

> The prediverticular state, the first stage of diverticulosis, was discovered by Marxer and described briefly about two years ago. . . . Part of the wall of the bowel, instead of showing its natural full curve of outline, appears fixed and contracted, with a profile of minute irregularities; this may be seen in only one side of the bowel, but if it involves the circumference there is the appearance of the stricture.

This statement appears to be erroneous, as George and Leonard, in their book "The Roentgen Diagnosis of Surgical Lesions of the Gastro-Intestinal Tract" (1915), recognized this appearance; for they say:

> Particularly along the descending colon and sigmoid one occasionally finds a peculiar serrated appearance. This may extend over several inches of the gut and is associated with more or less narrowing of the lumen. These serrations are small, close together, and with a rather sharp point, presenting at times a saw-tooth appearance . . . The serrations do not change in size or shape. Furthermore, palpation under the fluorescent screen usually shows us that the intestine is more or less like a rigid tube . . . One infers that the serrated appearance is due to inflammatory thickening and induration about the intestinal wall, secondary to the presence of diverticula, the diverticula themselves not being visible.

This type of case, the prediverticular state, formed almost 20% of Spriggs and Marxer's cases, and they mentioned that small diverticula could be frequently seen in or near these affected areas. My impression is that the incidence of this type of case is much less than 20% when compared with the cases of simple diverticula. Spriggs and Marxer hold that the recognition of this state alters the previous conception of diverticulosis, and suggest that some antecedent change in the bowel wall predisposes to the formation of diverticula, which had been thought to be due to passive extrusions of the intestinal mucosa through weak places in the wall of the bowel; and various agents of an infective type were suggested as possible predisposing causes. Lockhart-Mummery opposes this theory, stating:

> The evidence that I have obtained by a study of the early changes in the colon wall itself completely negatives this view and shows that the development of multiple diverticula is the primary stage and is unaccompanied by any inflammatory condition.

He shows sections demonstrating that these serrations in the prediverticular state are actually extremely minute diverticula projecting into, but not through, the muscular coats of the bowel, and that there is no evidence of inflammatory change. It is interesting to note that as far back as 1917 Carman and Miller showed a section of such a minute diverticulum separating the circular muscle fibres.

In discussing the differential diagnosis from an X ray point of view, Carman and Miller in 1917 said:

> . . . in the instance of carcinoma supervening upon diverticulitis, if rounded extra-luminal shadows were present, the lesion could not be distinguished from a benign diverticulitis, while if these shadows were not present the case would probably be regarded simply as one of carcinoma.

This brings under discussion the incidence of carcinoma supervening on diverticulitis. William Mayo suggested that irritation by hardened fæcal masses in the diverticula might be the cause of chronic irritation and precancerous change. His own figures suggest that carcinoma developing in diverticula is rare. In discussing diverticulitis and dealing with 1,918 recorded cases of diverticulosis and 1,259 cases of carcinoma of the colon, he refers to several cases in which carcinoma developed in a sigmoid in which the results of an ancient deforming type of diverticulitis were present. Mailer also apparently is opposed to this, for he says that it is impossible to say whether carcinoma develops *propter hoc* and not simply *post hoc*, and feels that the malignant process starts in the main lumen of the bowel and not in the diverticula, though the latter may be involved by extension.

My experience is that in a large percentage of the cases of carcinoma of the colon found by X ray examinations, no evidence of diverticula can be detected in the skiagrams; that in view of the fact that both lesions occur especially in middle or old age, one would expect many more to show the combination of the two lesions, carcinoma and diverticula, if there was any relation between them.

It is interesting to note also that in the other part of the alimentary tract where diverticula are common, namely, the duodenum, carcinoma is rare. Of course, it might be maintained that duodenal diverticula are not the same in ætiology or type as colonic diverticula; but one sees diverticula of the duodenum sometimes retaining fibrous tissue of a vegetable nature, which could act as an irritant in much the same way as a fæcolith in a colonic diverticulum. The radiological characteristics of carcinoma of the colon are usually such as to make it easy to distinguish carcinoma from diverticula, but at times it may be difficult or even impossible. Cases are occasionally encountered in which there is an actual obstruction by the mass due to the diverticulitis and peridiverticulitis; in such cases it may be quite impossible to express an opinion as to the cause of the obstruction. As a rule the defects due to carcinoma are more limited in length and have more even margins to them; they give much more the impression of masses invading, as it were, the barium shadow of the lumen, a lumpiness that is not characteristic of diverticulitis, in which the irregularity extends over a much greater length of bowel, and the narrowing has a spastic appearance with deformity of the diverticula due to inflammatory changes in the wall, often leading to the so-called "pallisading" of Spriggs and Marxer.

Though congenital diverticula of the colon have been described and apparently in rare cases do occur, the colonic diverticula we know are essentially acquired. The youngest patient mentioned by Mailer is over twenty years old; I have not met a single case at the Royal Alexandra Hospital for Children.

The ætiology has been discussed by Sir Charles Bickerton Blackburn, as well as the pathology; so that all that now remains for me to do is to show some slides illustrating the reason for left-sided pain and discomfort arising from disorders of the colon, and to illustrate certain features of diverticula of the colon and diverticulitis that have been discussed.

THE OCCURRENCE OF JUXTAARTICULAR NODULES IN AUSTRALIA.

By FRANK J. FENNER,[1]
University of Adelaide.

IN this paper the occurrence of two cases of juxtaarticular nodules is reported and discussed. As far as can be ascertained, these are the first cases recorded from Australia. One of the patients was a male Australian aborigine, apparently suffering from the endemic disease *irkintja*, and the other a white woman who was born in Scotland and had never lived in the tropics. The geographical incidence of the condition, and its pathology, ætiology and diagnosis, are considered, and its history and clinical characters are briefly reviewed.

[1] Assisted by a grant from the David Murray Scholarship Fund of the University of Adelaide.

DEFINITION.

We may define juxtaarticular nodules as:

> . . . subcutaneous nodules, rounded, solitary or agminate, situated in the neighbourhood of joints and bony ridges. Sluggish in growth, of firm and resistant consistency, they may exceptionally soften and end in a cicatrix which has no other characteristic than its juxtaarticular location. (Jeanselme, quoted from Hudson.[20])

Authors differ in their estimates of the frequency with which the nodules occur near the various joints. If we accept the idea that trauma plays a part in the determination of their site, it can be readily realized that there will probably be regional differences. Bernard,[3] in a recent review of the subject, arranges the following sites in the order of their frequence: elbow, knee, trochanter, external malleolus, anterior tuberosity of the tibia, head of the fibula, dorsal surface of the fingers, ulnar and tibial crests, acromion, sacro-coccyeal region and costal cage.

These nodules are usually about the size of a hazel-nut, but may grow as large as an orange. On the other hand, they may be much smaller, so that they are not obvious on inspection, but can be felt under the skin (Gutierrez[17]). In shape they are rounded or oval, and are usually hard, of cartilaginous or even woody consistency. Rarely they may become fluctuant and ulcerate, and this latter course seems to be more common when they are associated with syphilis.

The overlying skin is usually unaltered, but may in rare cases be smooth and shiny (da Fonseca[14]). The nodules lie in the subcutaneous tissue, adherent neither to skin nor deeper structures, although sometimes they are tethered to the periosteum beneath. Characteristically multiple, two, three, four and even up to fifty may occur in one patient. Their course is chronic; when the patients appear for treatment the nodules are usually of several months' and often of several years' standing. They inconvenience their owners little, being neither painful nor tender.

HISTORY.

According to Burnier,[5] juxtaarticular nodules were first described in 1778 in Guadeloupe, and in 1786 in San Domingo by a French physician, Bertin, who noted the relationship of the nodules to syphilis and yaws. In 1891, more than a hundred years later, the Brazilian physician Lütz mentioned them incidentally in a letter from Honolulu. Da Matta[29] has pointed out that Lütz also published an account of his findings in Brazil in the *Monatshefte für Praktische Dermatologie*, in 1892.

Sir William MacGregor,[27] in 1900, called attention to "a curious multiple tumour" found in the natives of New Guinea. In 1904, Steiner,[33] not knowing of MacGregor's work, described similar tumours in natives of Malaya. He studied them histologically, but came to no conclusions about their ætiology.

Unaware of these earlier publications, Jeanselme[22] described these tumours before the *Congrès Coloniale* and gave them the name of *"Nodosités juxta-articulaires"*, by which they are now known throughout the world. He[23] also pub-

lished a full clinical and anatomico-pathological account of the condition.

Hu and Frazier[21] have pointed out that Mackenzie[28] and Duckworth,[13] in 1883, and Fowler,[15] in 1884, published in England descriptions of conditions which were probably syphilitic juxtaarticular nodules.

Since 1900 an extensive literature has grown up on the subject. Most of this, although not available in Australia, is well summarized in *The Tropical Diseases Bulletin*.

Geographical Distribution.

The accompanying map (Figure I) shows the distribution of recorded cases of juxtaarticular nodules. The map is constructed from the information contained in Burnier's[5] article and *The Tropical Diseases Bulletin*. Where the authors have considered the lesions to be of syphilitic or frambœsial origin, I have indicated the fact. Where the observations are classed as "ætiology undetermined" the writers reporting the cases have either posited a mycelial origin of the nodes or have expressed no definite opinion on the subject.

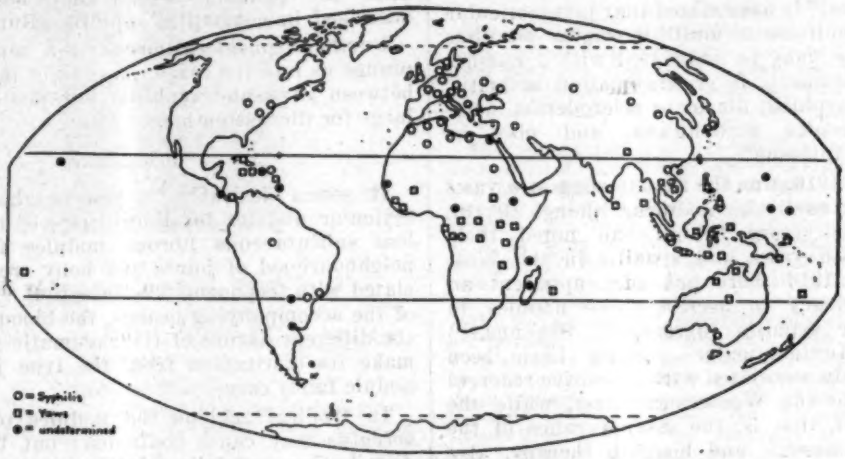


Figure I.
Map of world, showing distribution of juxtaarticular nodules.

The map illustrates the tendency for observers in temperate climates to link juxtaarticular nodules with syphilis, whilst those in tropical climates link them with yaws. As Hudson[20] has pointed out, there is a great disparity between the frequency of the affection in temperate and tropical regions; it is very rare in temperate climates and comparatively common (up to 1% to 2% of the population) in certain regions of the tropics. Nearly all the European reports, for instance, are of single cases, while many of the reports from Africa and Dutch East Indies describe up to one hundred cases.

The two cases described below are the first to be reported from Australia. Of these the case of the European female (Case II) almost certainly has a syphilitic ætiology. It is probable that the Australian aborigine (Case I) suffers from *irkintja*, which Hackett[18] has identified with yaws; but the differential diagnosis of late *irkintja* from late syphilis is not easy when, as in this instance, a reliable history is not available. However, Dr. C. E. Cook, Chief Medical Officer of the Northern Territory Administration, has seen several patients with juxtaarticular nodules. He writes:[9]

> . . . during my survey of aboriginals in North-west Australia in 1924 quite a number of cases (of juxtaarticular nodules) were noted. The commonest sites were in the region of the hip joint and the elbow joint. Quite a number of cases also were seen during the survey of aboriginals in the Northern Territory in 1925 and subsequent years during routine examinations of aboriginals by the Northern Territory Medical Service.

As Hackett[18] has pointed out, yaws is common and syphilis non-existent among these nomadic northern tribes.

Pathology.

The macroscopic and microscopic factors described by various authors differ considerably, and this difference seems to depend mainly on the different ages of the specimens examined. No sequence of changes according to the age of the lesion has yet been determined. Jeanselme in his original descriptions recognized three zones, the innermost showing degeneration, the outer inflammation, and the intermediate transition.

Hudson[20] has recently summarized the histopathological findings from the descriptions published since 1920, and we may quote his "composite histological picture":

> The tumours when cut across are seen to consist of hard greyish-white fibrous tissue, sometimes dotted with opaque yellow spots (Jeanselme), with very poor blood supply and occasionally areas of softening. There is no definite capsule. Microscopically, they consist of dense, compressed fibrous tissue, the fibrils twisting and overlapping, with numerous foci of chronic inflammatory mononuclear infiltration, lymphocytes, fibroblasts and plasma cells. Necrotic areas are occasionally seen. Such vessels as are present usually show a moderate obliterative thickening and perivascular infiltration with lymphocytes. Giant (Langhan's) cells may be found. There is no tubercle formation. The periphery of the tumour is loose connective tissue with perivascular round cell infiltration. The connective tissue towards the centre becomes hyaline and collagenous. This seems to be especially true in the larger nodules.

Jessner,[24] in a comprehensive summary of the cases associated with syphilis, points out that both clinically and histologically the lesions in the syphilitic cases show considerable deviations from other specific lesions.

Ætiology.

The ætiology of juxtaarticular nodules has been the subject of many controversies, and the problem is still unsettled. The earlier workers (Fontoyant and Carougeau,[16] in Madagascar, and Currie and Hollman,[10] in Hawaii) considered that the nodules were of mycotic origin. Their observations have not been confirmed, and it seems probable that their results were due to contamination.

Several writers (Joyeux,[26] Brault[4] and recently Stannus[31]) have stated that juxtaarticular nodules is a syndrome of multiple origin. Stannus states that they "may be associated with a number of diseases: rheumatic fever, rheumatoid arthritis, yaws, leprosy, syphilis, filariasis, scleroderma, acrodermatitis chronica atrophicans, and may be simulated by xanthoma".

Davey,[11] in 1916, was the first to suggest a yaws ætiology for juxtaarticular nodules, although Bertin, over a hundred years before, had noted their relationship with yaws and syphilis in the West Indies. Since 1916 there has been apparent an increasing tendency to ascribe these nodules to either yaws or syphilis (Jessner,[24] Stannus,[32] Hudson[20]). Juxtaarticular nodules have been almost invariably associated with a positive reaction of the blood to the Wassermann test, while the therapeutic test, that is, the disappearance of the nodules under arsenic and bismuth therapy, also supports the idea of a treponematous origin.

As indicated earlier, workers in tropical regions have recognized yaws as the ætiological factor, while those in temperate zones associate juxtaarticular nodules with syphilis. Hudson[20] has insisted that there has been a failure of writers to distinguish between ætiology and epidemiology. While considering juxtaarticular nodules as treponematous lesions, he thinks that the factors responsible for their frequent occurrence in yaws (in tropical regions) and their rarity in syphilis (in "civilized countries") are epidemiological; that is, the almost universal incidence, the childhood acquisition, and the almost total lack of treatment in the tropical regions.

Attempts to demonstrate treponemata in the nodules have met with a limited amount of success. Clapier,[6] van Dijke and Oudendal,[12] and Sobernheim,[32] have demonstrated spirochætes in nodule juice and in sections. By intratesticular injection of an emulsion of the nodule into a rabbit, and subsequent transfer of a lymph node of this animal to a second rabbit, Jessner and Rossiansky[25] obtained a specific lesion in the second rabbit. Hu and Frazier[21] were successful with the first generation of inoculated rabbits. Attempts to demonstrate the spirochætes in sections and by inoculation into rabbits were unsuccessful in the present investigation.

Jessner[24] has suggested that though the lesions occur in syphilitic persons they may not be syphilitic in nature. The few positive results mentioned above seem to be sufficient to prove the treponematous origin of juxtaarticular nodules; for spirochætes are hard to demonstrate in many tertiary lesions of yaws and syphilis.

Juxtaarticular nodules occur most commonly in men and women from thirty to fifty years of age, although they are met with in much older persons. The sex distribution is almost equal both in the yaws and syphilitic cases (Hu and Frazier[21]). In syphilis the nodules may very rarely appear as late secondary or early tertiary lesions; but they are usually late tertiary, often appearing many years after the primary sore. They have also been described in congenital syphilis (Burnier[5]).

Further consideration of the ætiology would plunge us into the vexed question of the relationship between yaws and syphilis; but this matter is too large for discussion here.

Diagnosis.

It seems desirable to reserve the term juxtaarticular nodules for those cases of multiple painless subcutaneous fibrous nodules found in the neighbourhood of joints and bony crests, and associated with treponematous infection. Consideration of the accompanying lesions, the blood serology and the different nature of the rheumatic nodule should make its distinction from the true juxtaarticular nodule fairly easy.

In tropical regions the nodules of *Onchocerca volvulus* may cause confusion; but their different distribution (rarely in relation to joints) and finally exploratory puncture will decide the diagnosis. Clapier[7] has given a good account of the differential diagnosis of these conditions. Burnier[5] has listed the conditions in tropical and temperate climates with which juxtaarticular nodules may be confused; if juxtaarticular nodules are kept in mind, however, the diagnosis is easy.

Report of Two Cases.

Interest in the diseases of the Australian aborigine has been quickened by the work of C. J. Hackett,[18] who has established the relationship between the *irkintja* of the Australian natives and the condition known as boomerang leg. He considers also that *irkintja* is identical with yaws, and thinks[19] that amongst aborigines many of the conditions diagnosed as syphilis were really yaws. In this paper I have assumed Hackett's identification of *irkintja* and yaws to be correct. The terms used below are those defined by Hackett.[18]

Case I.

C.J., a male Australian aborigine, aged about sixty-five years, from Horseshoe Bend, Northern Territory (latitude 25° S.), was examined at the Parkside Mental Hospital, Adelaide, South Australia.

History.

The patient was born and had lived the greater part of his life in native camps in Central Australia. Lack of an

interpreter made adequate history-taking out of the question; but he said that he had had *irkintja* when a young man and that he had had lumps on his knees for a long time.

FIGURE II.
The patient C.J. (Case I), showing juxtaarticular nodules over knees and left trochanteric region. A similar nodule was excised from over the right trochanter.

Examination.

The skin over the nose was rough and coarsely granular; but no scars were detected in the moist crease areas of the body.

The right tibia was straight and in the left one a very slight boomerang-leg deformity was present. X ray examination gave the following information: The right tibia was slightly bent in an antero-posterior direction; there was no increase in the antero-posterior diameter; there were dense cortical shadows; there were no abnormal shadows in the medullary cavity. The left tibia was definitely bowed anteriorly; the antero-posterior diameter was considerably increased; the cortical shadows were vague. The fibulæ were both slightly bent medially, and each had a small area of local periostitis in the middle of the shaft.

The reaction of the serum to the Wassermann test was strongly positive.

There were large painless swellings over both trochanters and both patellar regions. The appearance of this is well shown in the accompanying photograph (Figure II). That over the left knee was the largest, and was spherical and about eight centimetres in diameter. It had a smooth surface, had well-defined boundaries, felt hard, and was somewhat anchored to the underlying bone, the skin being freely movable over it. The rest were similar in character, but were not anchored to the underlying tissues. They were situated as follows: one, about five centimetres in diameter, over the lower part of the right patella and the head of the tibia; one, about three centimetres in diameter, over the left; one, about one and a half centimetres in diameter, over the right trochanteric region; and two small ones on the palm of the right hand, one over the middle of the volar surface of the first phalanx of the thumb, and one over the palmar surface of the second metacarpal bone. The nodules were quite painless and were not tender.

The nodule over the right trochanter was excised and examined histologically (Figure III). It was made up of very dense fibrous tissue containing few cells. The outer zone was composed of looser fibrous tissue with more cells and vessels. There was a little fibrous thickening around the arterioles; but no endarteritis or round-cell infiltration was present. A few small nerve fibres were seen in one section. No spirochætes could be found. No acid-fast bacilli could be detected in a section stained by the Ziehl-Neelsen method. A portion of the nodule was ground in sterile saline solution and injected into the testis of a rabbit. There was no reaction, and an examination of the testes for spirochætes some six weeks later proved fruitless.

Treatment.

A short course of neoarsphenamine therapy caused a slight regression of the nodes; but owing to difficulties of administration this treatment was not persisted in.

Comment.

The salient features of the case are the positive reaction of the serum to the Wassermann test, the slight boomerang-legs (radiographically) and the multiple symmetrical fibrous nodules.

Case II.

F.S., a female, aged fifty-three years, a native of Scotland, came to Australia twenty-five years ago. She had never lived near the tropics. She was examined at the out-patient department of the Adelaide Hospital.

History.

The patient had had five pregnancies, the first three being normal. The fourth baby died of "convulsions". The last pregnancy, eighteen years ago, resulted in a miscarriage at six and a half months. Her husband, who had served in the Great War, died eight years ago, at the age of fifty-two years, after several attacks of a right-sided hemiplegia. His blood gave a "doubtful weak positive" reaction to the Wassermann test, and his condition had been diagnosed as probable cerebral syphilis.

The patient had had painless lumps on various parts of her body for about thirteen years. The first ones noticed were three or four small nodules the size of peas on the front of the left wrist. These disappeared after two or three years and she then developed lumps on both elbows, over the right external malleolus, over both knees, both ischial tuberosities, and one on the palm of the right hand. The nodule on the right knee broke down and discharged after having been knocked about six years ago, and healed, leaving only a small scar. A similar ulceration of the nodule over the left knee, following repeated trauma, would not heal and brought the patient to the out-patient department of the Adelaide Hospital.

Examination.

The only abnormal findings were the multiple painless nodules, all of which were hard and had fairly regular surfaces. All were in the subcutaneous tissue and the skin was freely movable over them. They were situated as follows: two, about two centimetres in diameter, in the neighbourhood of the right elbow, one over the olecranon and fixed to the underlying periosteum and one over the lower end of the humerus; one, about one centimetre in diameter, over the left elbow, fixed to the periosteum of the ulna; one, about three and a half centimetres in diameter, over the right ischial tuberosity, and a similar one, four centimetres in diameter, on the left, both being freely movable; one small nodule, less than one centimetre in diameter, on the palm of the right hand.

There were a scar over the right patella and an ulcer over the left knee. The floor of the ulcer was covered with flabby granulations.

The reaction of the serum to the Wassermann test was strongly positive.

One of the nodules from the right elbow was excised and examined histologically (Figure IV). Macroscopically it was a firm, rounded non-encapsulated tumour, about the size of an almond, and on section had a homogeneous greyish-white appearance. Microscopically the nodule consisted of dense fibrous tissue, the fibrils twisting and overlapping and appearing in places as concentric rings around the vessels. There were small areas of recent hæmorrhage. The vessels were well formed and there were considerable perivascular accumulations of round cells. There were more cells scattered through the fibrous

tissue than in the preceding section (Case I). One section through the more superficial part of the tumour showed distinct thickening of the walls of the blood vessels and very abundant perivascular round-cell infiltration.

No spirochætes could be found in a section stained by Levaditi's technique. Intratesticular injection of an emulsion of the nodule into a rabbit gave negative results after ten weeks.

Treatment.

After a course of neoarsphenamine and bismuth therapy the nodules, when an examination was made nine months later, were found to have almost completely disappeared.

Comment.

The salient features of this case are the positive reaction of the serum to the Wassermann test, the multiple long-standing symmetrical fibrous nodules, and the response to antisyphilitic treatment.

DISCUSSION.

The diagnosis of *irkintja* in Case I is tentative. The bony changes were characteristic of a mild boomerang-leg deformity as described by Hackett.[18] The patient was an old man who had lived his life in aboriginal camps. Hackett has produced evidence which shows that in these circumstances *irkintja* is rife and syphilis non-existent. Hackett's identification of *irkintja* and yaws has recently been questioned (Black and Cleland[3]).

Amongst the Australian aborigines we have Hudson's epidemiological triad of universal prevalence, childhood acquisition and non-treatment of a treponematous infection. Hudson says that under these conditions the nodules should appear in the high percentages common in the tropics. However, this case is the first that has been seen amongst the Central Australians, although Cleland,[8] Hackett[18] and Basedow[1] have each examined large series of natives. The nodules seem to be more numerous in Northern Australia and are fairly common in New Guinea, where, it is said, the natives distinguish between yaws and syphilis and always associate juxtaarticular nodules with yaws.

Case II would probably fall into the category of the subcutaneous fibroid syphiloma of Parkes Weber.[34] It seems best, however, to consider this lesion as a type of juxtaarticular nodule. Recent work has tended to contradict Stannus's suggestion that the two groups of juxtaarticular nodules (yaws and syphilis) may become clearer as more and more of them are recorded with clinical and pathological findings. Most recent writers (Jessner,[24] Hu and Frazier,[21] Hudson[20]) consider that it is impossible to distinguish the juxtaarticular nodule of syphilis from that of yaws by histological examination. The sections described here support this opinion, although in Case II there were definite arterial thickening and perivascular round-cell accumulations which were absent in Case I.

Although many of the syphilitic cases are associated with other definitely syphilitic lesions (see Burnier[5]), it has been noted in several of the European cases that the patients were quite unaware of ever having had syphilis. The patient described as Case II falls into the latter class. Here the doubtful history of syphilis in the husband and the positive response of the blood to the Wassermann test make the diagnosis of syphilis probable.

SUMMARY.

1. Two cases of juxtaarticular nodules are recorded and described.

2. One patient was an aboriginal from Central Australia, the subject of *irkintja*, and the other was a syphilitic Scottish woman who resided in Adelaide, South Australia.

3. Microscopic sections of the nodules are described.

4. Attempts to recover spirochætes after inoculation into rabbits were unsuccessful.

5. The problem of juxtaarticular nodules in general is discussed and a map of the geographical distribution of the condition has been compiled.

ACKNOWLEDGEMENTS.

I have to thank Dr. H. M. Birch and Dr. A. Lendon for permission to publish these cases, and for their ready assistance, Professor A. E. Platt and Professor J. B. Cleland for help with the laboratory work, Professor F. Goldby for the photomicrographs, and Dr. C. J. Hackett for his continued interest and suggestions. Dr. C. T. Ch. de Crespigny has kindly read through the manuscript.

REFERENCES.

(1) H. Basedow: "Diseases of Australian Aborigines", *The Journal of Tropical Medicine and Hygiene*, Volume XXXV, 1932, page 177.

(2) R. Bernard: *"Les nodosités des saillies osseuses"*, *Bruxelles-Médical*, Volume VII, June 26, 1927, page 1033 (quoted by Hudson, *loco citato*).

(3) E. C. Black and J. B. Cleland: "Pathological Lesions in Australian Aborigines, Central Australia (Granites) and Flinders Range", *The Journal of Tropical Medicine and Hygiene*, Volume XLI, March 1, 1938, page 69.

(4) J. Brault: *"Note au sujet des nodosités juxta-articulaires chez les Indigènes"*, *Bulletins de la Société de pathologie exotique*, Volume IX, 1916, page 341 (abstracted in *The Tropical Diseases Bulletin*, Volume VIII, September 15, 1916).

(5) R. Burnier: *"Les nodosités juxta-articulaires et leurs rapports avec la syphilis"*, *La Presse Médicale*, Volume XLI, June 21, 1933, page 995.

(6) P. Clapier: *"Nodosités juxta-articulaires et tréponèmes"*, *Bulletins de la Société de pathologie exotique*, Volume XVI, 1923, page 553.

(7) P. Clapier: *"Les porteurs des kystes filariens (Onchocerca volvulus) et de nodosités juxta-articulaires en pays Toma (Région militaire de la Guinée)"*, *Bulletins de la Société de pathologie exotique*, Volume X, February, 1917, page 150 (abstracted in *The Tropical Diseases Bulletin*, Volume X, August 15, 1917, page 100).

(8) J. B. Cleland: "Disease amongst the Australian Aborigines", *The Journal of Tropical Medicine and Hygiene*, Volume XXXI, 1928, page 53.

(9) C. E. Cook: Personal communication, November, 1936.

(10) D. H. Currie and H. T. Hollman: "The Histological and Bacteriological Investigation of a Juxta-Articular Nodule in a Leper", *New Orleans Medical and Surgical Journal*, Volume LXXI, 1919, page 384 (abstracted in *The Tropical Diseases Bulletin*, Volume XV, March 15, 1920, page 180).

(11) J. B. Davey: "The Ætiology of Juxta-Articular Subcutaneous Nodules", *Annals of Tropical Medicine and Parasitology*, Volume IX, 1915, page 421.

(12) M. van Dijke and A. J. F. Oudendal: *"Voorkomen, bouw en oorzaak der Nodositès juxta-articulaires (van Jeanselme) bij Inlanders"*, *Geneesk Tijdschr v. Nederl-Indie*, Volume LXII, 1922, page 413 (abstracted in *The Tropical Diseases Bulletin*, Volume XX, April, 1923, page 288).

(13) D. Duckworth: "Case of Rheumatismal Cutaneous, Subcutaneous and Periosteal Nodules: Probable Syphilitic Taint", *Clinical Society Transactions*, Volume XVI, 1883, page 190.

(14) O. da Fonseca: *"Um typo particular de nodosidades juxta-articulares"*, *Boletim do Instituto Brasileiro de Sciencas*, Volume II, April 24, 1927, page 365 (abstracted in *The Tropical Diseases Bulletin*, Volume XXV, January, 1928, page 10).

(15) J. K. Fowler: "Subcutaneous Nodules Occurring in an Adult not the Subject of Rheumatism", *The British Medical Journal*, Volume I, January 19, 1884, page 107.

(16) M. Fontoyant and J. Carougeau: *"Nodosités juxta-articulaires, mycose due aux discomyces carougeaui"*, *Archives de parasitologie*, Volume XIII, 1909, page 583.

(17) P. D. Gutierres: *"Nodosités juxta-articulaires"*, *Archives of Dermatology and Syphilology*, Volume XII, 1925, page 159.

(18) C. J. Hackett: "Boomerang Legs and Yaws in Australian Aborigines", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Monograph 1, 1936, page 1.

(18) C. J. Hackett: "A Critical Survey of Some References to Syphilis and Yaws among the Australian Aborigines", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, May 30, 1936, page 733.
(19) E. H. Hudson: "Juxta-Articular Nodules in Euphrates Arabs", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXVIII, March, 1935, page 511.
(21) C. Hu and C. N. Frazier: "Study of Subcutaneous Nodules of the Juxta-Articular Type Observed in Five Cases of Syphilis in North China", *The Chinese Medical Journal*, Volume XLVII, 1933, page 364.
(22) E. Jeanselme: *Comptes rendus de la Société de médicine et d'hygiene coloniales*, 1904, page 15 (quoted by Guiterrez, *loco citato*).
(23) E. Jeanselme: *"Des nodosités juxta-articulaires observées sur indigènes de la Presqûile Indo-Chinoise"*, *Revue de médicine et d'hygiène tropicales*, Volume II, 1905, page 11.
(24) M. Jessner: *"Über syphilitische juxta-artikuläre Knotenbildungen"*, *Archiv für Dermatologie und Syphilologie*, Volume CLII, November 13, 1936, page 132 (abstracted in *The Tropical Diseases Bulletin*, Volume XXIV, April, 1927, page 311).
(25) N. Jessner and N. Rossiansky: *"Die Ergebnisse der Deutsch-russischen Syphilis Expedition, 1928"*, *Archiv für Dermatologie und Syphilologie*, Volume CLX, 1930, page 224.
(26) C. Joyeux: *"Contribution à l'étude des nodosités juxta-articulaires"*, *Bulletin de la pathologie exotique*, Volume VI, December, 1913, page 711 (abstracted in *The Tropical Diseases Bulletin*, Volume III, March 30, 1914, page 325).
(27) W. MacGregor: "An Address on Some Problems of Tropical Medicine", *The Lancet*, October 13, 1900, page 1055.
(28) S. Mackenzie: "A Case of Subcutaneous Nodules Occurring in a Patient the Subject of Syphilis, and with very Indefinite Connection with Rheumatism", *Clinical Society Transactions*, Volume XVI, 1883, page 188.
(29) A. da Matta: *"Primeiros casos de nodosidades de Lütz-Jeanselme no. Amazonas"*, *Brazil Medico*, Volume II, July 30, 1921, page 26 (abstracted in *The Tropical Diseases Bulletin*, Volume XIX, February, 1922, page 166.
(30) H. S. Stannus: "Yaws and Syphilis: A Critical Review", *The Tropical Diseases Bulletin*, Volume XXIII, February, 1926, page 82.
(31) H. S. Stannus: Comment in review of paper by H. Hopkins, *The Tropical Diseases Bulletin*, Volume XXIX, July, 1932, page 537.
(32) G. Sobernheim: *"Spirochätenbefunde bei den sogenannten Malayen fibromen"*, *Centralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten*, Volume XCIII, 1924, page 260.
(33) L. Steiner: *"Über multiple subcutane harte fibrose Geschwulste bei den Malayen"*, *Archiv für Schiffs- und Tropen-Hygiene, Pathologie und Therapie exotischen Krankheiten*, Volume VIII, 1904, page 156.
(34) F. P. Weber: "Chronic Fibroid Subcutaneous Syphilomata of the Legs associated with Chronic Peri-Urethral Induration of the Penis", *The British Journal of Dermatology and Syphilis*, Volume XXXII, 1920, page 173.

SOME ASPECTS OF PSITTACOSIS AND THE ISOLATION OF THE VIRUS.

By A. R. TREMAIN, B.Sc.

(From the School of Public Health and Tropical Medicine, the University of Sydney.)

History.

As the details, given by Sturdee and Scott (1930),[1] of the first recognized epidemic of psittacosis in Ulster, Switzerland, in 1879, are of interest, they are recorded here.

There were seven cases, with three deaths, in the household of some relatives of a physician named Ritter. Four of the people lived in the house, and of the other three, one came daily to clean the birds' cages. The owner of the house was a bird collector, keeping parrots, finches and exotic birds. A consignment had recently arrived from Hamburg, Germany. Of these, some were ill and one died, all the remainder being subsequently killed as suspect after the members of the household fell ill. Ritter described the disease as a "pneumo-typhus". The lung signs were at first those of bronchitis and later those of lobular pneumonia. Epistaxis occurred in no less than four cases. Ritter assessed the incubation period as nine to fourteen days, and found no evidence of human case-to-case infection.

Autopsies on the birds, conducted by Eberth, revealed no abnormality. The type of parrot is not stated; but shortly afterwards, in 1883, Eberth and Wolff examined some grey parrots from the Guinea Coast, in an endeavour to explain the immense mortality amongst consignments to Germany, and found micrococci present in great numbers.

The subsequent history reveals how observers of different epidemics have striven to establish the importance of parrots and similar birds in the production of the disease in man, and how the finding of a specific bacillus led to the establishment of a fallacious bacteriological hypothesis, which prevented the establishment of the true ætiology for a period of fifty years.

It was during the epidemics of 1892 and 1893 in Paris that Nocard isolated a Gram-negative, short, motile bacillus, pathogenic not only for parrots, but also for fowls, pigeons, mice, rabbits and guinea-pigs, from the bone marrow of the wings of parrots which died on the voyage from Buenos Aires four months before. He could not obtain any of the birds actually imported alive and presumed to be responsible for some of the cases in the first oubreak in Paris. The later discovery by Gilbert and Fournier, in 1896, of a similar bacillus in the heart blood of a patient suspected of having died from psittacosis, the isolation of a similar bacillus by Achard and Bensaude, and the introduction of the Widal reaction, increased the confusion in the diagnosis of cases of apparent Salmonella infection with psittacosis.

Since 1879 Sturdee and Scott have reported epidemics in Italy in 1897, and in Germany in 1882, 1896 and 1898. Only one case appears to have been recorded in America prior to 1917, when an epidemic occurred in Pennsylvania. A single case was reported in Pennsylvania in 1929. Although the epidemic of 1892 in Paris and the Italian epidemic of 1897 were caused by parrots coming from South America, no cases of the disease were reported in that country. In fact, the only evidence of the disease there, apart from the epidemic in 1929 and 1930, was one case in Brazil in 1904. In England, prior to 1930, seven cases of psittacosis were reported in 1914, 1924, 1926 and 1927.

Epidemiology.

It was not until 1929 and 1930, when pandemics occurred in many countries, that attention was concentrated on the disease and its true ætiology was discovered. Almost simultaneously, in April, 1930, Levinthal,[2] in Germany, and Coles[4] and Lillie,[5] in England, reported the presence of small ovoid or spherical bodies arranged in clusters and pairs, in microscopic preparations from virulent human and parrot materials. Bedson Western and Levy Simpson (1930),[7] in England, were the first to report the presence of a filterable virus in the organs of a parrot. Later, Bedson and others described the limited nature of its filterability.

The worst outbreaks affecting man appear to have occurred in America and Europe. Meyer *et alii* (1935),[10] state that since December, 1929, 189 cases and 14 suspected cases of psittacosis, with 40 deaths, were reported in the United States of America and in Canada. In 170 of these cases direct contact with shell parrakeets was proved. That infection of the aviaries in California had become considerable is shown by the fact that 70 cases occurred from exposure to locally bred birds, and shipments of the Californian birds to other places were responsible for 98 additional cases. These authors believe that the original stock for the Californian aviaries came from various countries, including Japan and Australia. Sturdee and Scott mention the Brazilian Amazon parrots as playing a part in the pandemics of America and Europe.

Levinthal (1935),[8] reports that during 1934 many human cases occurred in Holland and Germany. He quotes Fortner and Pfaffenberg as giving the number of human cases in Germany as nearly 150 during the first six months of 1934—almost three-quarters of the total number in 1930. In Germany, as in most countries, restrictions were placed on the importation of parrots and parrakeets in 1930.

Working in England, in April, 1934, Levinthal detected the virus in a shipment of Australian galahs and budgerigars, which arrived at Plymouth direct from Adelaide, and reported his results to the Commonwealth Health Department, at about the same time as Meyer, in America, was investigating a shipment of 200 shell parrakeets from Australia, caught about the environs of Adelaide, and forwarded to him as birds that had not been in contact with the infection. After receiving Meyer's report,[9][11] that psittacosis was present in these birds, the Commonwealth Health Department obtained the services of Dr. F. M. Burnet, who had recently been engaged in virus investigations overseas, to carry out a survey to determine the extent of the disease in Australian birds. He not only found that freshly caught birds of the parrot family, including the budgerigar, *Melopsittacus undulatus*, or shell parrakeet, from various States were infected, but also proved that human cases were occurring in Victoria. This confirmed the fact that psittacosis was an enzootic infection in our native birds.[12][13] In his report Keogh (1936),[15] who was working in association with Burnet, mentions that in four months eighteen human cases were investigated, and from the sputa of five patients the virus was isolated.

The American, European and Australian results indicate an incubation period, in man, of seven to fourteen days. The age distribution indicates a higher degree of infection in middle age. Of 64 patients in America, only three were under twenty years of age; of eighteen patients in Victoria, only three were under eighteen years of age; and of 104 patients in England, 60% were over the age of forty years. The incidence of human case-to-case infection was very low.

The Isolation of the Virus from a Bengalese Finch Imported into New South Wales.

That a bird, as far as is known, not previously reported as having been found infected, can be concerned in this disease is revealed by an investigation of an imported finch, found dead amongst a shipment from China on the steamship *Nankin*. It was received at the School of Public Health and Tropical Medicine, University of Sydney, on October 21, 1936, for examination for the presence of fowl "plague", and was described as a Bengalese finch. Birds of this description appear to be crosses between *Aidemosyne malabarica* and *Uroloncha* (*Munia*) *striata*, or of the latter with the Indian silver bill. They are hybrids that have lost the characteristics of the original types in the breeding, which is particularly prolific in the Orient.

As the bird had been dead for some time, the *post mortem* appearances were indefinite. Two white leghorn cockerels and two mice were inoculated from an emulsion of the spleen. The inoculation of the cockerels revealed an absence of fowl "plague". As one of the mice was found dead on the fifth day, with signs of putrefaction, the other mouse, apparently well, was killed. Smears made from the surface of the spleen and from the peritoneal fluid, stained by Castaneda's method, when examined microscopically, had the appearance of fine blue intracellular bodies, suggesting the possibility of Levinthal-Coles-Lillie bodies (hereinafter referred to as L.C.L. bodies). One of the two mice subinoculated from the spleen died twelve days later. The autopsy revealed an enlarged spleen, with a fibrinous covering which was also noticeable over the liver. Smears made from the surface of the spleen had microscopic appearances similar to those of the first preparations.

Of the following two mice inoculated from the spleen and liver, one, *H*, died eight days later. *Post mortem*, signs of pericardial exudate only were found. Smears from this revealed a number of comparatively large spherical bodies resembling micrococci. At first it was thought that these organisms had arisen from a secondary infection; but reference to two articles by Levinthal,[8] and Bland and Canti (1935),[16] gave sufficient information to indicate that these coccal forms could be the large forms of L.C.L. bodies. Closer examination of the films led to the detection of both large and elementary forms present in scattered cells. The presence of L.C.L. bodies in these smears was confirmed by Dr. F. M. Burnet.

Subsequent subinoculations in mice were continued until May 29, 1937, when a total of 81 mice had been used for about 30 passages, and the strain was lost. Those examined microscopically revealed the presence of mostly elementary forms of the L.C.L. bodies.

The results of the primary inoculation of the mice and the cockerels, together with the period of incubation in the mice early in the series, may indicate that the virulence was not of a high order, and that it was of a similar nature to that of the

Australian strains isolated by Burnet. It was enhanced by passage, so that some two months after the primary inoculation mice were dying after three and four days. At this stage attempts were made to test the effect of cold storage on the virulence of the virus in the spleen, without the addition of a preservative. From one of the mice dying on the third day after injection the spleen was removed and placed in a sterile test-tube plugged with cotton wool, and kept in the refrigerator at 4° C. for seven days. On removal from the refrigerator it appeared to be partly dried; but inoculation with it killed a mouse in three days, the autopsy revealing typical signs and microscopic appearances. Subsequent inoculations from this mouse gave positive results. Another spleen from a mouse that died on the sixth day was treated similarly. It was kept in cold storage for sixteen days, after which it appeared to be very dried; but inoculation into a mouse resulted in death in eleven days, typical signs being found *post mortem*. Meyer[10] mentions that desiccated bird spleens remain virulent at 5° C. for 277 days.

The Inoculation.

Before the *post mortem* examination, the bird and the mice were first dipped in a 5% solution of "Lysol", then pinned to sheet cork with "Gem" pins. The bird was then plucked.

The method adopted for the preparation of the inocula was to grind the tissue, spleen, or spleen and liver, in a sterile mortar, and make a suspension in five cubic centimetres of sterile physiological saline solution. The sterilization of the two-and-a-half-inch porcelain mortars and pestles was done in the autoclave. The injection was given intraperitoneally, usually in amounts of 1·0 cubic centimetre, although smaller quantities of 0·5, 0·25 and 0·1 cubic centimetre were used successfully.

The mice were kept in round glass jars, of 10·0 centimetres (four inches) in diameter, 15·0 centimetres (six inches) in height, with a depth of about 5·0 centimetres (two inches) of bran. These were provided with metal screw-on lids, perforated and covered with cheese muslin. The mice were fed daily with bread soaked in milk, and the jars were changed three times a week.

The Microscopic Examination.

The smears and impression preparations were prepared thinly on new, clean, grease-free slides, and allowed to dry in air. Following the early use of Castaneda's staining method, a modified method along similar lines to those used by Rivers,[18] Bedson[3] and Burnet[14] was employed.

The stains were made up as follows:

Stain A:

Phosphate buffer solution (pH 7·0)	95 cubic centimetres.
Formalin	5 cubic centimetres.
Löffler's methylene blue	10 cubic centimetres.

Stain B:

0·2% "Safranin O" solution	25 cubic centimetres.
0·1% acetic acid solution	75 cubic centimetres.

Stain *A* was applied to the unfixed film for three minutes. The film was then rinsed in distilled water and counterstained with stain *B* for thirty seconds. The film was then washed in distilled water, blotted with filter paper and allowed to dry in air.

Several workers have used Giemsa stain. It was found to be satisfactory for the larger forms of L.C.L. bodies, particularly in the smears from mouse *H*, and gave good permanent preparations.

For the staining of the sections of tissue, hæmatoxylin, eosin, Giemsa, and Heidenhain's iron hæmatoxylin stains were used.

The Pathological Examination.

The autopsy examinations revealed the signs described by MacNalty (1937).[17] In addition it has been noted that the appearance of the diaphragm suggested necrotic changes. In two mice pronounced necrosis of the liver was visible, numerous foci being seen throughout, and in one of these mice congested areas of hæmorrhage of a pneumonic type were noticed. From this mouse sections were cut from the liver, lung and diaphragm, after fixation in 5% formalin solution.

A histological examination was carried out by Dr. E. Ford. Generalized degenerative changes were found in the liver, with multiple foci of varying appearance, of poor staining and loss of cell characters, of granular débris, of polymorphonuclear leucocytic infiltration, and of mononuclear and lymphoid cells. L.C.L. bodies were visible in the cells of Kupffer, in the free phagocytes in the sinuses, and also in endothelial cells in the exudate on the serous surface. The lung displayed vascular engorgement, and lymphoid and mononuclear-celled infiltration of the alveolar walls.

The epithelial cells lining the alveoli were enlarged and many were free in the spaces. L.C.L. bodies were visible in the latter. In patchy small areas the alveoli were filled with erythrocytes. Pronounced desquamation of the lining epithelium of the bronchioles was noted. The muscle fibres of the diaphragm stained poorly, with patchy areas of hyaline degeneration and replacement by lymphoid and endothelial cells. Some fragmentation was seen, and a generalized cellular infiltration was present.

The Disease in Birds.

Keogh describes the symptoms of the acute infection as listlessness, ruffled feathers, semi-closed eyes, fits of shivering, loss of weight and atrophic breast muscles, laboured breathing due to mucus at the nasal orifices, and occasionally diarrhœa. If the bird dies, the autopsy reveals a few drops of mucus at the nasal orifices, a large, heavy, pale liver, occasionally studded with necrosis and infarcts, a slightly enlarged and congested spleen, and rarely some pneumonic patches on the lung. The virus may be demonstrated in the blood and organs; direct smears from the spleen usually exhibit L.C.L. bodies.

The percentage of birds suffering from the acute infection is usually small, and it is more often found in immature birds kept in crowded or unhealthy conditions. The more common latent infection is shown by enlargement of the spleen. The medium-sized spleens are more infective than the large ones. It appears that large spleens indicate that the bird has practically overcome the virus, and it is difficult to infect mice from them.

The Isolation of the Virus from Several Australian Parrots and Finches.

Following the isolation of the psittacosis virus from the Bengalese finch, examinations of Australian birds that died during transport to Sydney, or while in quarantine, have been carried out. The following have been found to harbour the virus:

Birds caught in New South Wales:

- Crimson Rosella (*Platycercus elegans*).
- Ringneck parrot (*Barnardius barnardi*).
- Cockatiel (*Leptolophus hollandicus*).
- Regent parrot (*Polytelis anthopeplus*).
- Eastern Rosella (*Platcercus eximius*).
- Port Lincoln parrot (*Barnardius zonarius*).
- Pale-headed Rosella (*Platycercus adscitus*).

Birds caught about Darwin, Northern Territory:

- Gouldian finch (*Poephila gouldiæ*).
- Long-tail finch (*Poephila acuticauda*).

The periods of incubation in the mice of the strains from these birds indicate virulence of a fairly low order, similar to that of the other strains isolated in Australia.

Further attempts to prolong the preservation of the virus in mouse spleen are being carried out on these strains in a similar manner to that given above, and also by the desiccation of mouse spleen after it has been ground up with sterile sand. To the present time, by the latter method, the virus has shown no appreciable loss of virulence after storage at 4° C. for three weeks.

Comments.

The fact that the psittacosis virus has been isolated from an imported Bengalese finch and from Australian finches is of interest, as it reveals aviary types of birds found harbouring this disease here, and adds to the source of infection already known to be considerable in our native birds.

That finches may become infected when placed in the breeding pens of budgerigars has been proved by Meyer.[10] In America two patients contracted the infection through contact with canaries. In England one case is reported through contact with a canary. In the Argentine cases have occurred through contact with thrushes and an Italian Cardinal bird. Levinthal,[3] in describing the German epidemic of 1934, states that the disease appeared generally in the form of family endemics, and the investigations revealed, with rare exceptions, the fact that the source of infection was not a sick parrot, but an apparently healthy budgerigar. He also states that the budgerigar is less susceptible to psittacosis than are most species of parrots, and frequently becomes an apparently healthy carrier of the virus.

Epidemics amongst humans have been reported in Victoria by Kennedy (1936),[20] and by Ross and Rabinov (1936);[21] yet no known cases have been reported elsewhere in Australia.

Laboratory diagnosis, to date, depends mainly on the isolation of the living virus from the bird or from the patient's sputum. Rivers advises that the blood is infective only when taken in the first three days, and is rarely available for diagnosis, since psittacosis is not suspected by the physician at this early stage. In describing the examination of the sputum he advises the intraperitoneal injection of mice, in triplicate, using 0·5 and 0·05 cubic centimetre of broth emulsion of the sputum. Any pneumococci present are responsible for the death of the mice within three days, and any surviving animals manifest signs of psittacosis from the fourth day onwards. Rivers and Berry (1931),[18] in a series of 60 sputa, conclusively demonstrated the virus in 14 cases, when it was collected from the fifth to the thirty-seventh day. In fatal cases the virus may be isolated from the lung. Burnet, in Australia, has confirmed the findings of Rivers.

At the present stage serological diagnosis is unsatisfactory. Although Bedson and Bland report satisfactory results with the complement fixation test, Keogh, in a personal communication, states that he has been unable to confirm their findings in a series of Australian cases. The complement fixation test was always inconclusive, and elicited no response in some cases proved by isolation of the virus. He suggests that possibly the less virulent Australian strains do not provide such satisfactory antigens in the test. Rivers and Schwentker (1934),[19] found that monkeys, recovered from psittacotic pneumonia, had an increased resistance to infection and possessed neutralizing antibodies in their sera; that the intravenous or the intramuscular injection of the living virus into monkeys and into humans was relatively harmless, and that repeated inoculations led to the appearance of neutralizing antibodies in the blood.

Summary.

1. From a bird imported from China and described as a Bengalese finch, psittacosis has been isolated by animal inoculation.

2. The strain appears to be of low virulence, comparable with that of the Australian strains isolated by Burnet. Virulence was enhanced by animal passage, so that the mice were dying on the third and fourth days some two months after the primary inoculation.

3. Infected mouse spleen may be preserved by cold storage, without preservative, at 4° C. for one week without loss of virulence, and for sixteen days with little loss of virulence.

4. Both large and elementary forms of the L.C.L. bodies have been detected.

5. It is believed to be the first isolation of this disease in New South Wales, and the first of its kind from a bird imported into Australia.

6. A list is given of Australian parrots and finches, caught in New South Wales and in the Northern Territory, found to harbour this disease.

Acknowledgement.

The author is particularly grateful to Professor Harvey Sutton, Director of the School of Public Health and Tropical Medicine, for the opportunity of being able to carry out this investigation. It was made possible by the arrangement between the Department of Agriculture, New South Wales, and the Commonwealth Health Department, for the examination of birds in quarantine.

References.

(1) E. L. Sturdee and W. M. Scott: Reports on Public Health and Medical Subjects of the Ministry of Health, Number 61.

(2) W. Levinthal: *"Die Ätiologie der Psittakosis"*, *Klinische Wochenschrift*, Volume IX, April 5, 1930, page 654.

(3) W. Levinthal: "Recent Observations on Psittacosis", *The Lancet*, Volume I, May 25, 1935, page 1207.

(4) A. C. Coles: "Micro-Organisms in Psittacosis", *The Lancet*, Volume I, May 10, 1930, page 1011.

(5) R. D. Lillie: "Psittacosis: Rickettsia-Like Inclusions in Man and in Experimental Animals", *Public Health Reports*, Volume XLV, April 11, 1930, page 773.

(6) S. P. Bedson and J. O. W. Bland: "Complement-Fixation with Filterable Viruses and their Antisera", *The British Journal of Experimental Pathology*, Volume X, December, 1929, page 393.

(7) S. P. Bedson, G. T. Western and S. Levy Simpson: "Observations on the Ætiology of Psittacosis", *The Lancet*, Volume I, February 1 and 15, 1930, pages 235 and 345.

(8) S. P. Bedson: "The Nature of Experimental Bodies in Psittacosis", *The British Journal of Experimental Pathology*, Volume XIII, February, 1932, page 65.

(9) K. F. Meyer and B. Eddie: "Psittacosis in Native Australian Budgerigars", *Proceedings of the Society of Experimental Biology and Medicine*, Volume XXXI, May, 1934, page 917.

(10) K. F. Meyer, B. Eddie and I. M. Stevens: "Recent Studies on Psittacosis", *American Journal of Public Health*, Volume XXV, May, 1935, page 571.

(11) J. H. L. Cumpston: *"La psittacose en Australie"*, *Bulletin de l'Office international d'hygiène publique*, Volume XXVII, July, 1935, page 1362.

(12) F. M. Burnet: "Psittacosis in Australian Parrots", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, December 8, 1934, page 743.

(13) F. M. Burnet: "Enzootic Psittacosis amongst Wild Australian Parrots", *Journal of Hygiene*, Volume XXXV, August, 1935, page 412.

(14) F. M. Burnet: "Laboratory Investigation of Suspected Psittacosis", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, March 14, 1936, page 363.

(15) E. V. Keogh: Report of the Federal Health Council of Australia, Ninth Session, April, 1936.

(16) J. O. W. Bland and R. G. Canti: "Growth and Development of Psittacosis Virus in Tissue Cultures", *The Journal of Pathology and Bacteriology*, Volume XL, March, 1935, page 231.

(17) A. S. MacNalty: Reports on Public Health and Medical Subjects of the Ministry for Health, Number 80.

(18) T. M. Rivers and G. P. Berry: "Psittacosis: Experimentally Induced Infections in Mice", *The Journal of Experimental Medicine*, Volume LIV, July, 1931, page 105.

(19) T. M. Rivers and F. F. Schwentker: "Vaccination of Monkeys and Laboratory Workers against Psittacosis", *The Journal of Experimental Medicine*, Volume LX, August, 1934, page 211.

(20) G. J. Kennedy: "Psittacosis in Man", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, March 14, 1936, page 366.

(21) F. C. H. Ross and L. Rabinov: "A Local Epidemic of Human Psittacosis in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, March 14, 1936, page 364.

SOME OBSERVATIONS ON THE USE OF PROTAMINE ZINC INSULIN IN THE MANAGEMENT OF DIABETES MELLITUS.

By EWEN DOWNIE, M.D., M.R.C.P.,

Physician to Out-Patients and Officer in Charge of the Diabetic Clinic, Alfred Hospital, Melbourne.

ALMOST from the time of introduction of insulin into medical practice the idea of prolonging its action and delaying its absorption has attracted the attention of experimental workers. The need of a combination of insulin which would be more sustained in its effects than is the insulin of everyday practice should be apparent to any physician with knowledge of the treatment of *diabetes mellitus*.

Considerable difficulty is experienced in maintaining the blood sugar content of some patients within normal limits, and frequent injections of insulin may be necessary, often at inconvenient hours. This is exemplified by those patients who, after an injection of insulin, experience a quick fall of blood sugar content to a hypoglycæmic level, followed in a few hours by an equally sudden rise to abnormal heights. In some instances the patient suffers from hypoglycæmic reactions without warning, and may pass rapidly from a state of apparently normal health into coma and have no knowledge of this event. So, on theoretical grounds, a slower and more evenly absorbed insulin, the action of which would be spread over many hours, seems to possess obvious advantages in such cases, and also would be of assistance to many of those patients whose condition necessitates constant administration of insulin.

In recent years various attempts have been made to achieve this object; amongst these may be mentioned the efforts of Leyton, who sought to suspend crystalline insulin in oil, and of Clausen, who injected a vasoconstrictor substance together with insulin. Neither of these methods met with any pronounced success. Lately attention has been directed to the production of a more or less insoluble compound of insulin which, because of the time taken in its disintegration, would be absorbed slowly. At the present time this idea has met with greater success than any other, and credit is due to Hagedorn for the first such preparation of practical value. After prolonged experimentation he discovered that insulin could be combined with various monoprotamines, and he finally selected for his purpose one obtained from the rainbow trout. After considerable work on the most satisfactory proportion of protamine and insulin in combination protamine insulin was produced; and this substance has appeared under the commercial title of "Insulin Retard". Hagedorn investigated the possibilities of this substance on a selected group of patients, and observed them for more than a year before advancing his claims. He found that the action of insulin when combined with protamine was much prolonged, and that the sudden fall of

blood sugar occasionally observed in diabetic patients after the injection of insulin did not occur. Hypoglycæmic symptoms developed more slowly and less frequently in these cases, but were apt to be of longer duration. His patients felt better and were less tired. He found protamine insulin of great value in certain surgical complications of diabetes. He also observed that in many cases the total daily use of insulin could be lowered by as much as 15%. He found that the number of injections could be reduced often to one per day; but he noted that it was not possible to control all cases by the use of protamine insulin, and in these separate injections of ordinary and protamine insulin were necessary.

A further improvement in the efficacy of this compound was made by Scott, who showed that the addition of small quantities of zinc to protamine insulin resulted in the formation of a substance the action of which was even more prolonged. Such a combination of protamine insulin with zinc possessed the additional advantage that it was more stable chemically and could be kept under ordinary conditions for many months. Protamine insulin, being relatively unstable, can be kept at full potency for only a few weeks. Careful work by Scott and others has shown that, as far as can be ascertained, the injection of minute quantities of zinc into the human body over long periods of time is followed by no detrimental effects.

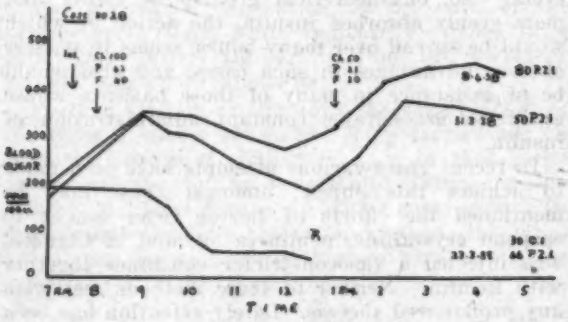


FIGURE I.

Protamine zinc insulin has now become a commercial preparation, and appears likely to displace the original protamine insulin as a therapeutic agent. Reports from authoritative clinics abroad, where this substance has been in use for over two years, indicate the value of this compound in the management of diabetes.

In February, 1937, Rabinowitch reported his results in 132 cases in which protamine zinc insulin was used. In only eight cases did he fail to control the diabetic condition. Of the remaining 124, 121 were controlled by one injection per day, and only three required two injections daily. After an experience of over 1,200 cases, Joslin hails the introduction of protamine zinc insulin as marking a new era in diabetic treatment, and reports difficulty of stabilization in only ten cases in this series. Up to the present time British literature is much more guarded in its comments upon this new compound, although Lawrence states that he considers that "the usefulness of zinc protamine insulin is established", and expresses a view that "all but the most exceptionally severe cases can be maintained in good health by one injection a day either of zinc protamine insulin alone or with added soluble insulin".

Time has not yet permitted a proper appreciation of the value of these compounds in treatment, and there is much to be discovered before their benefits can be fully assessed. It is quite apparent that in the use of a slowly absorbed insulin, individual variations in the rate of absorption are likely to be observed in different patients. It is probable that the bulk of the injection itself plays some part in the rate of absorption, and that an injection of 80 units will have a different effect from that of two injections of 40 units made simultaneously in different situations. The rate of absorption and of digestion of food, which is a matter of individual variation, must also merit attention in any attempt to provide a patient with extraneous insulin which will be effective for approximately twenty-four hours. In other words, the adjustment of the diet and the dosage of insulin are individual matters, and each patient's response must be separately studied before stability can be achieved.

My personal experience in the use of these new preparations has been limited to protamine zinc insulin; and during the past six months I have had the opportunity of observing its effects upon a group of 32 patients (Table I).

The number of patients is too small and the time of observation too short to allow of dogmatism; but it seems to me, despite some difficulties which I have experienced, that we are now possessed of a type of insulin which will prove of value in the future.

Of this group of 32 patients, 11 had not been treated with insulin previously, while 21 had been receiving ordinary insulin for a varying period of time (from some months up to ten years). It is advisable to consider the behaviour of these two groups separately.

Of the 11 patients who had not previously received insulin, nine patients can be considered to be in a satisfactory state, one is not satisfactorily stable, and in one case the use of insulin has been discontinued.

In the 21 cases in which treatment with insulin had previously been carried out, 13 patients are satisfactorily controlled on protamine zinc insulin alone, four are satisfactorily controlled on protamine zinc insulin with the addition of a small dose of ordinary insulin, the condition of three is unsatisfactory, and in one case the use of protamine zinc insulin has been discontinued.

Proceeding now to a consideration of some details of these groups, I shall discuss first those cases in which no previous treatment with insulin had been undertaken. Broadly speaking, two methods may be used to stabilize an untreated patient: either he may

be placed upon a basal or lower than basal diet, both diet and insulin being gradually raised until a satisfactory state of control is attained, or the patient may be given his total caloric requirements at the commencement of treatment and the insulin may be adjusted to his needs. The latter method, which has been my practice for some years when using ordinary insulin, has been applied in all the cases in this series. The patient is accommodated in hospital; but unless some definite indication exists he is not confined to bed. He is encouraged to exercise, and if possible to continue his ordinary daily work, using the hospital as a boarding-house. The patient is thus stabilized for his usual life and not for an artificial existence in hospital surroundings. The total caloric requirements of the diet are estimated; to begin with, it is arranged so that approximately two-tenths of the total carbohydrate for the day is given in the morning meal, three-tenths in the midday meal, four-tenths in the evening meal and one-tenth before retiring to bed. Usually treatment is commenced by the administration of equal amounts of protamine zinc insulin and ordinary insulin in two separate injections twenty to thirty minutes before the morning meal, no further insulin being given during the day. The amount of the initial dose depends upon the patient's condition and upon the fasting blood sugar content, and may range from 10 to 30 units of each type of insulin daily. The effect of treatment is gauged by the quantitative examination of at least four urinary samples in the twenty-four hours; the fasting urine (that obtained between the passage of the overnight sample and the consumption of the first meal) and samples obtained one to two hours after the three principal meals of the day. After two or three days the insulin dosage is raised or lowered according to the urinary findings, and an attempt is made to increase the dosage of protamine zinc insulin and to lower the dose of ordinary insulin until the latter reaches vanishing point. It has been my experience up to the present time that frequent alterations in the dose of protamine zinc insulin are apt to cause confusion, as it seems to take a few days for the full effect of any change to become apparent. After the patient has ceased to need ordinary insulin, it may be found necessary to alter the distribution of the carbohydrate food throughout the day and to distribute it evenly between the three meals or to add more carbohydrate to the supper before retiring.

Thus in Case IV the patient, after discharge from hospital, showed pronounced glycosuria in the evening, and raising of the insulin dosage produced hypoglycæmic symptoms during the night. Reversion to the former insulin dose and transfer of 15 grammes of carbohydrate from the evening meal (6 p.m.) to the supper (9 p.m.) were sufficient to restore stability.

Another patient in this group (Case XVIII) failed to reach stability by any variation of diet until the time of the injection of protamine zinc insulin was altered to one and a half hours before the morning meal. This procedure was followed by a clearing of sugar from all urine tests throughout the day.

TABLE I.

Case No.	Age in Years.	Sex.	Duration of Diabetes.	Previous Diet.					Previous Administration of Insulin, in Units.				Present Diet.				Partition of Diet.				Protamine-Zinc Insulin Units.	Ordinary Insulin Units.
				Carbohydrate. (Grms.)	Protein. (Grms.)	Fat. (Grms.)	Total Calories.	Partition of Diet.	Morning.	Midday.	Night.	Carbohydrate. (Grms.)	Protein. (Grms.)	Fat. (Grms.)	Total Calories.	Breakfast.	Lunch.	Dinner.			
I	7	F.	14 years	168	62	102	1,838	2/5	12	0	8	165	69	102	1,854	1/5	2/5	2/5		10	4
II	54	F.	1 year	150	70	150	2,330	2/5	12	0	8	150	70	120	1,960	1/5	2/5	2/5		16	
III	24	F.	11 years	125	75	90	1,610	1/5	16	nil	16	127	78	87	1,603	1/5	2/5	2/5		30	
IV	10	F.	4 weeks		nil					nil		198	86	128	2,288	1/5	2/5	2/5		38	
V	57	F.	3 years		Quali tative					0		125	70	130	1,950	1/5	2/5	2/5		30	
VI	47	F.	12 years	80	60	120	1,640	even	20	0	0	80	60	120	1,640		even	2/5		30	
VII	49	F.	9 years	160	80	150	2,430	2/5	18	0	14	200	100	175	2,775	1/5	2/5	2/5		12	
VIII	62	M.	10 years	182	80	140	2,268	2/5	22	0	12	193	93	177	2,737	1/5	2/5	2/5		24	
IX	8	M.	3 months	78	68	147	1,907	1/2	17	0	15	190	70	110	2,030	1/5	2/5	2/5		24	
X	63	M.	4 years		Quali tative			1/4		nil		110	70	120	1,800		even			nil	
XI	10	M.	14 years	177	68	100	1,867	2/5	15	0	10	198	86	128	2,288	1/5	2/5	2/5		22	
XII	50	M.	2 years		Quali tative			1/5		nil		260	123	193	3,269		2/5			30	
XIII	33	M.	6 weeks		nil			2/5		nil		175	85	165	2,525	1/5	2/5	2/5		10	
XIV	44	F.	7 years	80	60	120	1,640	even	60	0	15	125	68	100	1,672	1/5	2/5	2/5		44	
XV	7	M.	6 years	192	68	87	1,823	1/5	30	0	30	192	68	87	1,823	1/5	2/5	2/5		40	
XVI	30	M.	6 months		Quali tative			2/5		nil		200	100	175	2,775		even			40	
XVII	38	M.	2 years	90	90	150	2,410	2/5	16	0	12	175	90	150	2,410		even			26	
XVIII	41	M.	1 year	175	70	150	2,410	1/5	16	0	12	175	90	150	2,410	1/5	1/5	2/5		36	
XIX	29	M.	6 years		Quali tative			2/5		nil		308	123	193	3,461		even			38	
XX	67	M.	15 years	125	70	130	1,950	1/5	20	0	20	125	70	130	1,950	1/5	2/5	2/5		28	
XXI	62	M.	2 years		Quali tative			2/5		nil		150	70	120	1,960		even			36	
XXII	15	M.	2 years	175	100	130	2,270	2/5	20	0	20	150	70	120	1,960	1/5	2/5	2/5		24	
XXIII	28	M.	2 years	175	70	130	2,150	1/5	10	0	5	175	100	130	2,270		even	2/5		24	
XXIV	19	M.	6 months	200	100	150	2,550	2/5	20	0	20	175	70	130	2,150		even			40	
XXV	43	M.	4 years		Quali tative			2/5		nil		200	100	150	2,550	1/5	2/5	2/5		12	
XXVI	14	M.	3 months		nil					nil		150	90	150	2,310	1/5	1/5	2/5		36	
XXVII	18	M.	4 years	250	110	120	2,520	2/5	35	0	35	150	90	150	2,310	1/5	2/5			40	
XXVIII	46	M.	6 years		Quali tative			1/5		nil		250	110	120	2,520		even			26	
XXIX	65	F.	6 years	110	70	120	1,800	2/5	30	0	30	150	80	120	1,960	2/5	1/5	2/5		15	10
XXX	59	F.	10 years	175	90	160	2,500	1/5	15	0	15	125	80	120	1,800	1/5	2/5	2/5		44	16
XXXI	20	F.	3 years	100	75	150	2,050	2/5	25	0	25	110	70	120	1,800	1/5	2/5	2/5		30	
XXXII	45	F.			Quali tative			even	20	0	20	140	80	160	2,360	1/5	even	2/5		44	
										nil		150	90	150	2,310		2/5			40	
												150	70	120	1,960			2/5		30	

From time to time some patients in this group have shown transient glycosuria, which may be due to variation in the rate of absorption from day to day.

The accompanying table (Table II) serves to illustrate the stabilization of an untreated, severely diabetic patient with protamine zinc insulin.

The patient in Case XVI, a male, aged thirty years, had had diabetes for about six months, during which period he had lost 6·35 kilograms (one stone) in weight. When he was examined on November 26, 1937, his height was 165 centimetres (five feet six inches), his weight was 45·5 kilograms (seven stone two pounds), his fasting blood sugar content was 0·38%, and large amounts of acetone and diacetic acid were present in his urine. His condition has remained satisfactory ever since his discharge from hospital four months ago (Table II).

One patient in this group (Case V) is not properly controlled. She left hospital in quite a satisfactory state and returned to her home, 100 miles from Melbourne, where she has remained ever since. Shortly after her return she commenced to show sugar in her urine, and this state has continued for several months. Whether she is not carrying out her dietary treatment with precision or whether her behaviour to protamine zinc insulin has altered, I cannot say.

In Case X the use of protamine zinc insulin was soon discontinued because of the most intense local allergic reactions, a response which this patient had previously given to all available brands of soluble insulin.

Turning now to a consideration of the second group of 21 patients previously under treatment with ordinary insulin, I must emphasize the fact that, at the time of transition, satisfactory stability had been achieved in 18 while three were not adequately controlled.

It is interesting to note that the average dose of ordinary insulin in this group was 38 units per day. After the change to protamine zinc insulin the average daily dose became 33 units per day, which represents an average reduction in dosage of 13%. As I have already mentioned, 13 of these patients are now well controlled on protamine zinc insulin alone, while four require a small dose of ordinary insulin in addition. Three patients are not satisfactorily controlled, and one has abandoned the use of protamine zinc insulin and returned to ordinary insulin.

A number of patients in the group were not placed in hospital, but were permitted to lead an ambulatory life under close observation. With few exceptions the method of transfer from ordinary insulin to protamine zinc insulin was similar to that advised by Joslin: the morning dose of ordinary insulin was given as protamine zinc insulin, and the total remaining insulin for the day was given as ordinary insulin simultaneously with protamine zinc insulin before the morning meal. At the same time the diet which, according to my usual practice for patients taking ordinary insulin twice daily, was divided into sections of two-fifths, one-fifth and two-fifths, was changed to a distribution of approximately one-fifth, two-fifths and two-fifths. Urinary observation was carried out as mentioned before, the patients reporting the results of urine testing each day. At intervals of two to three days the protamine zinc insulin dosage was increased and the ordinary insulin dosage decreased until the patient was taking protamine zinc insulin alone. Considerable variation in the time taken to regain stability was observed, the shortest interval being seven days, the longest seven weeks.

In Case XI the patient was a boy, aged ten years, who for some months had taken 15 units of ordinary insulin in the morning and 10 units of ordinary insulin in the evening. He was rapidly transferred to 22 units of protamine zinc insulin in the morning, and his urine was completely sugar-free within a week, despite the fact that his diet was simultaneously raised by over 300 calories.

On the other hand, in Case VII the patient, who had been stable for months on 18 units of ordinary insulin in the morning and 14 units of ordinary insulin in the afternoon, experienced great difficulty in becoming stabilized on protamine zinc insulin alone. After seven weeks he eventually became completely sugar-free on 12 units of protamine zinc insulin *plus* eight units of ordinary insulin in the morning. It is interesting to note that his total insulin requirements have fallen from 32 units to 20 units per day.

At the present time I know of no explanation as to why it should be so easy to transfer some patients and so difficult to transfer others; and on more than one occasion I have recalled Joslin's advice with some regret: "Never attempt to teach

TABLE II.

Date.	Sugar Content of Urine.				Weight in Kilograms.	Units of Insulin.		Diet.
	Fasting.	At 9.30 a.m.	At 2 p.m.	At 7 p.m.		Protamine Zinc Insulin.	Ordinary Insulin.	
	%	%	%	%				
29/11/1937	4·0	4·0	4·0	4·0	45·0	—	—	
30/11/1937	6·0	3·0	2·0	2·0	—	20	20	2,775 calories, consisting of 200 grammes of carbohydrate, 100 grammes of protein, 175 grammes of fat.
1/12/1937	3·0	3·0	2·0	2·0	—	20	20	
2/12/1937	2·0	2·0	2·0	2·0	—	20	20	
3/12/1937	2·0	2·0	1·0	0·4	—	25	25	
4/12/1937	2·0	2·0	2·0	0·6	—	30	20	
5/12/1937	2·0	0·6	0·2	1·0	—	30	20	
6/12/1937	1·0	2·0	0·2	0·6	46·4	30	15	
7/12/1937	0·6	0·6	0·2	0·1	—	35	10	
8/12/1937	2·0	0	0	0	—	40	10	
9/12/1937	0	0	0	2·0	—	40	5	
10/12/1937	0·6	2·0	2·0	3·0	47·3	40	0	
11/12/1937	0·2	0·2	0	0·2	—	40	5	
12/12/1937	2·0	0·6	1·0	1·0	—	40	0	
13/12/1937	0·2	0	0	0	—	40	0	
14/12/1937	0	0·2	0	0	48·2	40	0	
22/12/1937	Sugar absent in all tests.				—	38	—	
12/ 1/1938	Sugar absent in all tests.				51·8	34	—	
30/ 3/1938	Sugar absent in all tests.				53·6	26	—	

an old dog, or a dog with established habits, new tricks."

In Case III the patient had suffered from diabetes for ten years. She was satisfactorily stabilized on 16 units of ordinary insulin twice a day, changed to 30 units of protamine zinc insulin within a fortnight and maintained a reasonable stability for four weeks; and then for no apparent reason she became gradually more and more unstable. Despite the subsequent administration of 40 units of protamine zinc insulin together with 30 units of ordinary insulin, her condition remained unstable, and she demanded a return to her former insulin régime. After a short period on 30 units of ordinary insulin twice a day her stability was regained, and her insulin dosage slowly returned to 16 units of ordinary insulin twice a day. Notwithstanding careful investigation, I was unable to detect any reason for this loss of tolerance.

Case XIV, on the other hand, provides an example of the value of protamine zinc insulin. This patient, after six weeks of careful observation in hospital with ordinary insulin, 60 units in the morning and 15 units in the evening, was quite unstable and suffered from hypoglycæmic attacks. Freedom from sugar was eventually achieved after some weeks with 40 units of protamine zinc insulin before the morning meal. Her response was of some interest because of the fact that at one stage her condition was unstable on 44 units of protamine zinc insulin and five units of ordinary insulin in the morning, and became quite stable on 40 units of protamine zinc insulin *plus* seven units of ordinary insulin. She has had only one reaction since beginning on protamine zinc insulin four months ago.

The response of the following two patients to protamine zinc insulin appears to me to be extraordinary, and I am still experiencing difficulty in obtaining a satisfactory state of stability.

The patient in Case I, a female, aged seven years, developed diabetes during 1936. She was easily stabilized on a diet of 168 grammes of carbohydrate, 62 grammes of protein and 102 grammes of fat per day, with 12 units of insulin in the morning and eight units in the evening. In September, 1937, she was transferred quite satisfactorily to 20 units of protamine zinc insulin before the morning meal. She progressed well for two or three months, and then she began to show signs of insulin reactions at intervals during the day. Lowering of the dose to 18 units for more than three days caused heavy glycosuria; raising of the dose to 20 units for more than a few days caused signs of insulin reaction which would persist for hours at a time. No variation in the distribution of the carbohydrate food throughout the day caused any alteration in this state of affairs. After some weeks she appeared to settle satisfactorily again on 18 units. Since February of this year, however, the same unbalanced state has been apparent. Thus, in one day, all four urine tests will reveal heavy glycosuria, with 18 units; the next day two or three tests will be clear; the next day signs of reaction may be noticed. Lowering of the insulin to 17 units causes persistent glycosuria. At another time the fasting and after-breakfast tests will be clear one day and loaded the next, and so on. To me there appears no sensible explanation of this child's behaviour to alterations of one unit in the daily dose. After weeks of vainly attempting to vary the distribution of food to match the bizarre effect of the insulin, I have decided to attempt to regain stability with protamine zinc insulin and ordinary insulin in conjunction. She has not been in hospital during the past three months, but the parental management of the diet and insulin is good, and I have no reason to suspect that these factors are in any way responsible for her strange reaction to protamine zinc insulin.

The patient in Case XXVIII, a male, aged eighteen years, developed diabetes in 1934, and maintained a reasonable state of stability on a diet of 250 grammes of carbohydrate, 110 grammes of protein and 120 grammes of fat, with 25 units of insulin in the morning and 20 units in the evening; but he was subject, at rare intervals, to sudden hypoglycæmic attacks without warning, and for this reason I attempted to transfer him to protamine zinc insulin. He has now been under observation in hospital for twelve weeks, and has been unstable all that time.

After some weeks, during which I slowly increased the dose of protamine zinc insulin and ordinary insulin in the mornings to 40 units of protamine zinc insulin and 30 units of ordinary insulin, he showed pronounced glycosuria throughout the twenty-four hours for days at a time. Then he began suddenly to develop hypoglycæmia before lunch. His blood sugar at this stage was at a very low level; and yet frequent examinations of the urine revealed the presence of sugar, suggesting some lowering of the renal threshold. I then placed him on 50 units of protamine zinc insulin for one week, with the result that his blood sugar remained at a persistently high level for the whole of the day. Increase of the dose of protamine zinc insulin to 80 units for one week resulted in his blood sugar attaining a higher level still (Figure III). I then decided to start from the beginning once more, and gave him 15 units of protamine zinc insulin and 10 units of ordinary insulin before the morning meal. To my amazement, he began to have reactions again before lunch, and although I have not yet had the opportunity of obtaining serial estimations of his blood sugar content since, his fasting blood sugar content is now 0·22%, only 0·02% higher than when he was taking 80 units of protamine zinc insulin. His general condition is quite good; he is at work at an office all day, and he has lost a few pounds in weight, but his health seems satisfactory. No variation of his diet appears to influence his behaviour, his urine is always loaded with sugar; and what I am to do with him, short of returning him to his former doses of ordinary insulin, I do not know.

Shortly after commencement of treatment with protamine zinc insulin, several patients in this series complained of feeling on the verge of insulin reaction for hours at a time, and this despite the fact that all urine tests throughout the day contained large quantities of sugar. In those cases in which I have had an opportunity of estimating the blood sugar content during this period, I found it invariably to be well above the hypoglycæmic level. In every case the feelings were transient and disappeared after a few days. At present I cannot offer any explanation for this occurrence.

It has been a common experience among the patients in this series that injections of protamine zinc insulin are nearly painless; and almost every patient who has had experience of the old and the new insulins remarks on the difference.

In Case VI the patient suffered persistent discomfort from local reactions to four different brands of soluble insulin, and this continued for over two years. The change to protamine zinc insulin caused an immediate and permanent cessation of local reactions.

Up to the present time I have not had the opportunity of observing the effects of protamine zinc insulin on the diabetic undergoing surgical procedures. It is probable that when time permits the use of protamine zinc insulin would be of advantage in maintaining the blood sugar of such a patient at a desirably low level.

Earlier reports on the use of protamine zinc insulin emphasized the fact that it was contraindicated in any condition in which rapid action of insulin was desired, and so it was not advocated in diabetic coma. However, Rabinowitch has recently contradicted this statement, and has shown

that in the treatment of coma by the early injection of large doses of protamine zinc insulin a rapid and prolonged fall of blood sugar and a satisfactory reduction of ketosis are obtained. I await an opportunity to try this in practice.

Finally, it would be unwise, if not foolish, for me to deliver any dogmatic conclusions based upon my present limited experience of protamine zinc insulin. Its behaviour is different from that of the ordinary insulin with which we are familiar, and I am of the opinion that many of my difficulties have arisen from my own inexperience. I am convinced that it has a definite value in the management of *diabetes mellitus*, but that further knowledge and experience of its action are necessary before its place in treatment can be determined.

Reports of Cases.

A CASE OF EXTREME HYPERSENSITIVITY TO INSULIN.

By REG. S. ELLERY, M.D.,

AND

P. GUY REYNOLDS, M.B., B.S.,

Melbourne.

W.C.B., a single man, aged twenty-eight years, was admitted to hospital on March 18, 1938, for a course of hypoglycæmic shock therapy. He was of asthenic habitus, like "yonder Cassius", reticent and introverted, somewhat despondent, yet with an irritability which quickly flared into anger. He complained that he could not eat his food, not because he had no appetite nor because he suffered from any gastric discomfort, but because the thought of food in his stomach troubled him so much that he was impelled to vomit. He thus ate only a few morsels at mealtime, and even this small amount was taken only after coercion. At night, a prey to acute hunger, he would go to his mother's kitchen and literally stuff himself with whatever food, cooked or uncooked, he could find. He would eat quickly and ravenously, as one whose sole bliss was eating; and when his hunger was appeased and his belly up-blown with his gluttony, he would go outside the house and vomit. More often than not, he would then return to the kitchen and go through the same ritual again, until, exhausted by excess, he would go off to sleep.

His previous history showed him to have been a good scholar, and he had gained an academic degree with distinction. But he was always shy and found it difficult to mix with his fellows. He had no real interest in games or in hobbies. During his undergraduate days his family had fallen upon adversity and the patient had been compelled not only to live frugally, but to eschew any outside interest which might have offset his introspective tendencies. His mother, who is highly strung, has an oversolicitous manner towards the patient, who himself has had no contact with or betrayed any interest in the opposite sex and who at the age of twenty-eight showed signs of sexual immaturity and aplasia.

At the time of his coming into hospital his illness had been apparent for about two years. Shortly after its commencement he had been sent to England in connexion with his work, but had soon been compelled to relinquish this and enter a nursing home, in which he stayed for some months, with little benefit. He returned to Australia early this year, terribly weak and emaciated and with no thought of returning to work. In fact, he showed not only an indifference to his work, but an alarming lack of interest in things outside himself. His behaviour was compulsive: it was not a "surfeit of the sweetest things" which turned his stomach sick, but the detumescent relief of tension which vomiting brought about; and with this "gastric orgasm" considerable feelings of guilt were associated. His wish to get well was superficial. His emotions were blunted. He was indifferent to the anxiety and financial embarrassment he was causing his family. He was dishonest and secretive and uncooperative. At times he was definitely antagonistic to treatment, and said he felt competent to outwit the doctor who attempted to cure him. Though his compulsion held his reason captive, his perception was clear and his ingenuity considerable. For subconscious reasons the path of righteousness was forsaken for the path of regression, the patient's behaviour being a suicide compromise in the face of difficulties which appeared insurmountable; and on account of the schizoid features presented in his behaviour it was decided to treat him by insulin shock.

Physically the patient at the time of admission to hospital was extremely emaciated with a pellagroid dermatitis on the dorsum of both hands, and œdema of the ankles. Examination of the urine revealed no abnormality. The systolic blood pressure was 80 and the diastolic blood pressure 48 millimetres of mercury. In view of his poor physical state it was decided to commence treatment with a low dose of insulin, to increase the dose cautiously, and, in the initial stages of the treatment, to terminate the hypoglycæmia short of the onset of coma. Vitamin B_1, in the form of intramuscular injections of "Benerva", was administered in order to increase the resistance to insulin. As there was no response to the initial dose of 10 units, the dose was increased on the second day of treatment to 15 units. Two and three-quarter hours after the injection he was flushed, sweating freely, drowsy and confused. The hypoglycæmia was terminated three hours after the injection by the administration of 100 grammes of glucose by mouth. The same dose of insulin was given on the third day of treatment. Three hours after the injection the patient was just able to drink the glucose solution. On the fourth day, one and a quarter hours after the injection of 15 units, he became comatose. The corneal reflexes could just be elicited, and the plantar response was bilaterally extensor. The hypoglycæmia was immediately terminated by the administration of 200 grammes of glucose by the nasal tube, and 50 cubic centimetres of a 33% solution of glucose intravenously. At 6 a.m. on the following day he again became very drowsy and passed urine into the bed. He was able to be roused, however, sufficiently to drink the glucose solution. On the sixth day of treatment, two hours after the injection of 10 units, the hypoglycæmia was terminated at the stage of drowsiness and slight sweating by the administration of 200 grammes of glucose by mouth. As the patient appeared to be passing into coma subsequent to the taking of the glucose, further glucose was administered half an hour later by the intravenous route. On the seventh day, one and a quarter hours after the injection of five units, the patient became comatose. The corneal reflexes could not be elicited, and the plantar response was bilaterally extensor. Because of the early onset of coma, the dose of insulin was reduced on the following day to two units; but the patient again passed into coma about one and a quarter hours after the injection. On the ninth day the dose was further reduced to one unit, yet coma, with the characteristic neurological findings, developed two and a half hours after the injection. On the following day 0·5 cubic centimetre of distilled water was injected as a control. Half an hour after the injection the patient remarked, "I don't think I'll get all wet today", and later, "I'm not going off today. I'm not even drowsy; I'm trying to induce it [that is, drowsiness], but it won't come". On the eleventh day coma was again induced by a dose of one unit. During the succeeding days the patient's resistance to insulin increased, with the result that by the fifteenth day the dose had been increased to five units. Three and a quarter hours after the injection a severe epileptiform seizure occurred, followed by a prolonged period of apnœa, necessitating artificial respiration. The patient, however,

quickly responded to the injection of adrenaline and the intravenous injection of glucose solution, and was awake five minutes after the occurrence of the seizure. On the twentieth day he had another seizure, which occurred during the passage of the nasal tube, three and a quarter hours after the injection of three units of insulin. Treatment was terminated on the twenty-fourth day, as it was considered inadvisable to proceed further, in view of the uncertainty of response and the danger of severe "after-shock" as a result of the patient's persistent and secret vomiting.

CASE OF ARACHNOIDITIS OF THE CEREBELLO-PONTINE ANGLE, CAUSING TRIGEMINAL NEURALGIA.

By A. E. COATES,
Melbourne.

H.B., a male, aged fifty years, was admitted to hospital in October, 1936, complaining of pain on the right side of the face for twelve months. The pain started in the region of the angle of the mouth and radiated laterally to the right eye and the forehead, and to the right occipital region. The pain was "jagging" and severe, and came on suddenly, lasting from three to twenty-four hours, and being only slightly relieved by aspirin, phenacetin and caffeine. Intervals of freedom from attacks were of some weeks' duration.

He had an injection of alcohol into the fifth nerve in April, 1936, and as a result he had a "dead feeling" on the right side of the face. The injection relieved the pain for about two weeks only, when it returned in as severe a form as before. He complained also of twitching of the right lip for ten months; but this was not related to the pain, and would occur at any time and almost every day. The sight in the right eye had been failing for about ten years, and at the time of admission to hospital he could see only a shadow with his right eye.

Examination revealed poor visual acuity in the right eye; the right pupil was larger than the left and reacted very poorly to light and accommodation. The right corneal reflex was absent. Anæsthesia to pin-point was noticed on the right side of the face in the region of the eye, nose and mouth. He could not feel a touch with absorbent cotton and could not distinguish between heat and cold in this region. The right palpebral fissure was narrower than the left, and the skin of the forehead on the right side could not be raised. Test of the deep reflexes gave no information, and neurological examination of the trunk revealed no abnormality.

X ray examination of the skull revealed disease of the ethmoidal sinuses, otherwise no abnormality. Neither the blood nor the cerebro-spinal fluid reacted to the Wassermann test. Results of the examination of the cerebro-spinal fluid were normal.

The right eye was myopic and affected with disseminate chorioiditis. There was no papillœdema.

At this time the patient improved owing to his absorption in arranging Christmas decorations. He was discharged with a diagnosis of possible cranial nerve arachnoiditis; but there was thought to be a large functional element in his pain.

In June, 1937, he was readmitted to hospital. The pain had become worse, and was localized more in the frontal region of the face. He had since developed a buzzing sensation in the right ear and had become deaf recently in that ear. His memory for recent events was poor and he had become very irritable. He had also noticed difficulty in speaking. He could not think of the correct word to say, even though he knew what he wanted to say. Furthermore, he was certain that his head had become larger, a larger size of hat being necessary.

Examination revealed a right facial paresis and ptosis and right pupil larger than the left and of sluggish reaction as previously noted. His reflexes were normal. Examination of the fundi revealed no papillœdema. The right eye was almost blind. Nystagmus was noticed, being more pronounced on looking to the left. Anæsthesia over the right side of the face was noted, as previously. There was nerve deafness of the right ear. There were no cerebellar signs. It was decided to explore the right posterior fossa.

An occipital flap was raised under local anæsthetic. In the cerebello-pontine angle, adhesions were present between the fifth, seventh and eighth cranial nerves. These were separated with a small hook. The fifth nerve was divided by a tenotome. No tumour was detected. After all the adhesions had been divided, the dura was replaced and the scalp sutured together. A tortuous artery was seen lying on the eighth nerve. This was gently tucked into a sulcus of the cerebellum.

The patient made a rapid recovery, and was relieved of all trigeminal pain and twitchings. The buzzing in the ear also disappeared.

Reviews.

A HISTORY OF WOMEN IN MEDICINE.

DR. KATE CAMPBELL MEAD, author of a monumental volume on the history of women in medicine, practised for many years as a physician in Connecticut.[1] She gradually developed an absorbing interest in the achievements of medical women, and so began what has been the main study of a lifetime. She was encouraged in her researches by Dr. Welch and Dr. Kelly, of the Johns Hopkins University, and by the late Sir William Osler. After years of fascinating and painstaking research in many of the greatest European libraries, she unearthed an astounding body of little-known and unpublished matter which had remained unnoticed in numbers of Latin and Greek manuscripts and which had been but rarely mentioned in any modern books. As the fruition of incessant labours, involving correspondence and often personal interviews with many physicians of her sex scattered through many countries of the world, she has written the first of two large volumes which will give women the credit due to them for the inestimable worth of their work in medicine.

Dr. Mead's first volume, now before us, tells the story of medicine in Egypt and Assyria, then proceeds to that of Greece and Rome, and then to that of mediæval Europe. The last sections of the book deal with the medical history of various countries until the opening of the last century.

The author's main aim, naturally, has been to stress the important, though unrecognized, rôle which women have played in this drama. Few of us, though interested in medical history, can possibly be aware, before reading this great work, of what vital interest and importance the woman doctor has been through the ages. It is likely, indeed, that the patience and capacity for sympathy of women, combined with their maternal instincts, make them a force in the fight against disease which our modern world does not appreciate, thought our ancestors from the earliest days appear to have done so. It would seem that there have always been women, sometimes queens little less than goddesses, sometimes priestesses, who have devoted their lives to the relief of suffering.

The great lack of feminine names in ancient and even comparatively modern medical literature has been largely due, says Dr. Mead, to the errors of copyists or of printers who mistook a feminine noun or adjective for a masculine. Thus the change of an *a* to an *e* or an *us* has made important differences in medical writing and has cheated many an able woman of her meed of praise. In the eleventh century, for instance, some careless scribe wrote "Trottus" for "Trotula", and so, according to Osler, he deprived a worthy woman not only of her renown, but of her identity and a chair.

[1] "A History of Women in Medicine from the Earliest Times to the Beginning of the Nineteenth Century", by K. C. Hurd-Mead; 1938. Haddam: The Haddam Press. Medium 8vo, pp. 585, with illustrations. Price: $6.00 net.

The amount of labour which Dr. Mead has expended upon her book may be judged by the fact that after 35 years of practice in America she buried herself in the British Museum, in which, for some two years, she busied herself in a hunt for authentic information. This work entailed the reading of original manuscripts written in Latin, Greek and many other languages, augmented by manuscripts from many other libraries. She states rather modestly that she hopes on the completion of her second volume to have covered her subject "with something approaching adequacy". If the second book is as painstakingly and interestingly written as the first, and provides her readers with such a mass of well-documented and authoritative information, the author need have no fears of the manner in which she has handled her subject. The book is not free from *errata*, but these are of minor importance and mainly typographical. The illustrations are uniformly excellent and many of them have not previously appeared. Dr. Mead is to be congratulated upon a fine piece of work; "Women in Medicine" should be read by all lovers of medical history and all women graduates.

ARTIFICIAL FEVER.

DR. CLARENCE A. NEYMANN, associate professor of psychiatry at Northwestern University, has written a book on the production of artificial fever by physical means.[1] He is the man who originated this form of therapy. This book, which supplies a long-felt want, will be invaluable to those who desire to treat patients by means of "electropyrexia". The work is well presented, written in a clear and concise manner, conservative in its claims and free from exaggeration.

Early chapters are devoted to the history, ancient and modern, of the use of heat in the treatment of disease. Fever is shown to be one of the natural defensive mechanisms of mammals against attack by parasitic micro-organisms. The temperature which is lethal to a micro-organism need not be reached in the human body; heat brings many other factors into play, for example, massive antibody formation, vigorous action of leucocytes, increased metabolism and more rapid circulation. It matters little whether fever is produced by the body itself or by physical agents.

In the chapters which deal with the physiology of hyperpyrexia, the difference between the actions of internal and external heat is described, the dangers and limitations of external heating are mentioned, and the warning that the rectal temperature, normally higher than the axillary, may be made lower by the application of external heat, is given. The means of production of the pyrexia and the possible ranges of temperature are described, together with the effects of high fever upon the blood cells and the circulation, on perspiration and sodium chloride excretion, on the urine and on the pulse and respiration. Neymann states that the pulse rate should not exceed 160 per minute when the higher temperatures are induced. Discussing perspiration, he says that 4,500 cubic centimetres of sweat are excreted during a period of treatment lasting eight hours; this amount contains 45 grammes of sodium chloride—an enormous loss to the body compared with a normal average excretion of about seven grammes daily. An increase in almost all its constituents occurs in the blood, due partly, but not entirely, to the decrease in blood volume. The metabolic rate increases about 7% for each rise by 1° Fahrenheit temperature above normal.

The author discusses the evolution and technique of electropyrexia, and states that he has used all methods of the electrical production of fever. His present practice is to use an air-conditioned cabinet and produce fever by electromagnetic induction. The cabinet was designed by himself and Holmquest. As the heart rate is dependent upon the temperature of the skin, the temperature of the cabinet must be comparatively low.

The diseases cured and lessened and those unresponsive to this treatment form the subject-matter of the latter half of the book. Syphilis in all its forms, and especially syphilis of the central nervous system, multiple sclerosis, *chorea minor*, corneal ulcer and gonorrhœa are conditions in which, according to Neymann, good results may commonly be obtained by the use of induced pyrexia. Patients with selected forms of asthma and arthritis are also said to have benefited. Other diseases in which occasional improvement has been manifested are *Brucella abortus* infections, meningococcal meningitis, rheumatic carditis and Parkinson's disease.

Of selected patients suffering from *dementia paralytica*, 80% recover after treatment; of institutional patients 30% recover, while a further 40% manifest decided improvement. Sixty *per centum* of patients suffering from *tabes dorsalis* obtain relief; in this disease the pains are mitigated or vanish and there is usually a return of bladder and rectal function and a decrease of the ataxia.

Both primary and secondary syphilis are apparently cured in a short time by a combination of heat and chemotherapy, but Neymann issues a warning that this combined therapy is not as yet the treatment of choice of early syphilis. Its final value will not be determined until at least twenty years have elapsed. Multiple sclerosis, treatment of which with heat is pregnant with difficulty, improves in approximately 70% of cases. In *chorea minor*, according to Neymann, "artificial fever is the only therapy which offers a prompt and efficacious cure". Neymann treated 25 patients, of whom 22 recovered, the average time spent in hospital being sixteen days. Recurrences affected 6% of these patients.

Hyperpyrexia is also claimed to be of use in the atrophic forms of arthritis, 10% to 15% of patients being greatly benefited, and between 30% and 40% obtaining some relief. About 15% of all patients suffering from gonorrhœa are not helped by electropyrexia, owing chiefly to the fact that the infecting organism is a resistant strain. For all other cases of this disease the treatment is claimed to be a specific.

To patients afflicted with Parkinson's disease following an attack of lethargic encephalitis, hyperpyrexia has brought relief; but in cases of *paralysis agitans* due to organic lesions of the mid-brain, it has, as might be expected, been found useless. It is well to remember that a latent tuberculous infection may be activated by heat.

The book ends on a note of caution against the employment of hyperpyrexia in numerous diseases which from their nature could not be expected to be influenced by fever.

FORENSIC MEDICINE.

IT is seven years since the appearance of the previous edition of "Glaister's Medical Jurisprudence and Toxicology". This, the sixth, has been re-edited and partly rewritten by the surviving Professor John Glaister.[1]

The familiar scheme of the treatise still survives, much new material has been added, and what is new is good. The Ruxton case has supplied a basis for new methods of investigation, and frequent reference is made to it in the book. The section on toxicology has been brought up to date. The dangers of many of the new drugs, so lavishly advertised, will be evident on perusal of these pages. The legal responsibility for death under anæsthesia is fully discussed. It is to be regretted that more attention has not been called to deaths after the use of local anæsthetics.

This book can be recommended with confidence not only to senior students, but to all practitioners, who might with benefit keep it not in their consulting rooms, but by their firesides. It will be found full of interesting information which can be absorbed with ease.

[1] "Artificial Fever Produced by Physical Means: Its Development and Application", by C. A. Neymann, A.B., M.D., F.R.S.M.; 1938. London: Baillière, Tindall and Cox. Baltimore: C. C. Thomas. Super royal 8vo, pp. 209, with illustrations. Price: 27s. net.

[1] "Glaister's Medical Jurisprudence and Toxicology", edited by J. Glaister, M.D., D.Sc.; Sixth Edition; 1938. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 760, with 107 illustrations and eight plates. Price: 25s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 10, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

MEDICAL MEETINGS.

THE medical profession has heeded well the scriptural adjuration of "not forsaking the assembling of ourselves together": of making many meetings there seems to be no end.

For many medical practitioners in the metropolitan centres, and particularly those who serve their professional brethren in medical politics, it has for long been a nice problem how to fulfil their obligations with respect to medical meetings and at the same time maintain something better than a mere bowing acquaintance with their families, obtain an occasional evening's recreation, and guard against that loss of interest in the humanities which renders the medical man a one-sided person, incapable of sustaining conversation in any field other than that of medicine.

The complexity of modern medicine renders it difficult to arrange meetings of general appeal, and there are good grounds for the suggestion that the monthly meetings of the Branches of the British Medical Association in the several States should show first consideration for the interests of the general practitioner. Specialists may be trusted to look after themselves; they have their own societies, both within and without the British Medical Association.

Moreover, attendance at medical meetings involves for the general practitioner, after a long day of visits and evening consulting hours, greater effort than the specialist is as a rule required to exert. Hence when the practitioner, wearied by his twenty or thirty visits a day, conscientiously endeavours to avail himself of the educational value of medical meetings, every effort should be made to divest the gatherings of the tiresome features which are apt to beset them.

The ideal medical meeting is that in which well-chosen subjects are presented in a concise and stimulating manner and discussion is frank and free, so that all participating feel at the end of the evening that they can say with Dr. Johnson: "Sir, we had a good talk."

Medical meetings as we know them take the form either of the presentation of a paper dealing with some aspect of medical science or the demonstration of interesting and important clinical features in patients assembled for the purpose. If for the moment, and for the sake of brevity, we designate the former type of meeting "scientific" and the latter "clinical", we hope we shall not play into the hands of the cynics who would see in the distinction an index of the disparity between scientific and clinical medicine. The "scientific" meetings, however, can be made too scientific and little can be expected in the way of discussion if the subject be ultra-academic. In that event the atmosphere becomes more that of the lecture room than of a meeting in which the speaker is inviting comment on his remarks. That the average travelogue should be admitted to the syllabus of scientific meetings is more than doubtful.

Clinical meetings of moderate size, stripped of much of the formality attendant on the monthly meetings of the Branches, appeal to many men in general practice, since they provide more intimate contact with their colleagues and greater opportunity for a "good talk"; but many clinical meetings have been spoiled and discussions rendered stilted and futile by lack of preparation on the part of those entrusted with the duty of presenting the case records of the patients to be considered. How

frequently has it been observed that the clinician in charge of a particular patient seems unfamiliar himself with the clinical facts and cannot present them in their proper sequence. See him delve one disjointed statement after another from the chaotic scribble of an out-patient's card, or make himself heard at intervals over the shuffling of the leaves of the clinical notes of a patient from one of the hospital wards!

Some men are better endowed than others with the faculty of demonstrating; some can sift the wheat from the chaff in a lengthy clinical record with greater facility than others; but all can avoid the evil of unpreparedness. Lack of preparation is a poor compliment to the meeting and engenders an atmosphere of listlessness, out of which little profitable discussion is to be anticipated.

And medical idiom! Surely to find clinical vernacular tiresome is not to be pedantic! Some of the clinical expressions in everyday use which figure largely in the vocabularies of speakers at clinical meetings, when translated, are very ridiculous. When it is stated that the patient experienced pain in the "right upper abdomen", is it not pertinent to inquire of precisely how many *abdomina* the patient is possessed? To say that such-and-such a man has a "negative Wassermann" implies that a "Wassermann" is something with which we are all endowed, carrying, like the electron, a positive or a negative charge.

The habit of calling the patient the "case" has been condemned repeatedly, but apparently to little purpose. We hope that this is an impropriety of diction only or, to put it more bluntly, speech that is slovenly, and that it does not imply the outlook which regards every patient merely as a "case", with no economic, temperamental or human background. Men of high, even of eminent, standing in the profession persist in this solecism, and when we hear such a one say, "This case had its leg in plaster for three weeks", we feel constrained to cry, as Talleyrand is reputed to have said of Napoleon, "How unfortunate that so great a man should have been so badly brought up!"

The scholarship of an Osler, the erudition and oratorical gifts of an Allbutt, or, not to go beyond our own immediate orbit, the liquid eloquence and artistry of expression of a Felix Meyer, are not to be expected of the average medical man. They are Olympian attributes. But the rank and file of medical men might reasonably be asked to cultivate something better than the current jargon which seems to have become established as a clinical dialect.

Current Comment.

REACTIONS TO BLOOD TRANSFUSION.

THE recent practice of using stored blood for transfusion to patients makes the study of reactions to blood transfusion a matter of ever-increasing importance. B. Fantos, L. Seed and E. Schirmer[1] have made an attempt to classify these reactions. They admit that their classification is largely based on theoretical considerations, but suggest that it may help to bring about a better understanding of such reactions as may occur, even when great care in the procedure is exercised. One possible cause is the employment of apparatus which has not been perfectly cleaned. The authors advise the use of transparent rubber tubing and the scrupulous cleansing of other apparatus, followed by repeated rinsing in sterile distilled water and subsequent sterilization. The donor must be free from transmissible diseases; a history of any manifestations of allergy should be sought and, if obtained, investigated. Everything possible should be done to prevent hæmolysis of the blood during storage. The use of isotonic anticoagulant solution, the avoidance of severe agitation of the blood, and the earliest possible storage of the blood in a refrigerator, at a temperature of approximately 4° C., are advisable. The authors have discovered—and this is an important finding—that blood stored for more than one week is apt to contain enough dissolved corpuscles to give a higher percentage of reactions than blood kept for a shorter period. Great care in typing is, of course, always important.

The authors, in common with others, have found that careless heating of the blood has been responsible for some reactions. They prefer to avoid this risk entirely; there is no need to heat the blood, provided the infusion is given, as it should be, drop by drop. They emphasize the danger of "speed shock". A weak myocardium may be overwhelmed by too rapid an administration of blood into the veins. The occurrence of febrile reactions may be anticipated in patients who are manifesting or are recovering from elevations of temperature. The authors believe the routine injection hypodermically

[1] *Archives of Pathology*, July, 1938.

of morphine sulphate, in a dose of 0·01 gramme, when the infusion is started, may prevent a thermic reaction. Allergic reactions may occur, even when the donor is not an allergic subject. Another danger is a possible lytic tendency of the patient's serum, not sufficiently intense to be revealed by cross-matching. Whenever icterus or subicterus is present, a hæmolytic tendency in the patient should be suspected and the physician should be reluctant to introduce foreign red blood cells into the circulation. In such cases serum alone is better tolerated.

EPIDEMIC DIARRHŒA AMONGST THE NEW-BORN.

W. H. Best reports that an epidemic of diarrhœa occurred in July, 1934, amongst new-born babies in one of the New York City hospitals and a very serious outbreak of enteritis occurred.[1] Seventy-two were infected, that is to say, 18% of the babies in the nursery. The mortality rate was 45%. Bacteriological investigations and necropsy examinations gave no clue to the nature of the epidemic. So far as Best could ascertain, about eight references to a similar condition have appeared. From July, 1934, to the end of December, 1937, Best investigated 27 further outbreaks in 19 different hospitals. Fourteen hospitals were affected by a single outbreak; three had two; one had three; and another had four. In the hospitals which experienced multiple epidemics, the time intervals did not indicate any relationship between the first and later outbreaks. There was no evidence of any seasonal or climatic predilection. Best followed the post-natal course of 5,082 babies born alive in the hospitals concerned. Of this number 750 contracted the disorder—a morbidity rate of 14·7%. Of the babies attacked 356 died. Thus the mortality rate was 7% and the case fatality rate 47·5%.

The symptomatology of the infection was interesting. It was noted that an infant, seemingly healthy and thriving, became unduly drowsy, and, if awakened, gave a short feeble cry. There might be a slight elevation of temperature. Loose, watery, yellowish stools were passed containing no mucus, blood or pus. Less often the colour was yellowish-brown or greenish. Occasionally the child vomited; distension of the abdomen was frequently seen. Watery stools of increasing frequency led to loss of the infant's weight even to the extent of a pound in twenty-four hours. The patients assumed the aspect of dehydrated, marantic infants in a state of shock. Death sometimes supervened in one or two days, or the disease ran a course of from five to seven days before recovery or death. In some of the fatal cases the patients contracted bronchopneumonia. The disorder was apparently limited to new-born babies and at no time did it spread to older children in the ward. No adult attendants in the nurseries became infected, nor did children becoming ill at home infect older members of the family. After the initial infection the spread in the nursery was rapid. This and the further fact that infants contracted the disorder in as short a time as one or two days after admission to a nursery containing infected babies suggested that the incubation period was very short. In one instance, however, the child became ill twenty days after discharge from hospital. The majority manifested signs of the disease between two and six days after presumed exposure. The outbreaks were not completely controlled until the maternity sections in the hospitals were closed to fresh admissions, the babies discharged and the quarters renovated. As in many hospitals supplementary feedings were given to the infants, pathways for infection were open both to breast-fed babies and to those artificially nurtured. Hence there was no marked difference in the incidence of the disease amongst these two classes. No common ætiological agent could be discovered. A variety of organisms, such as *Bacillus coli* (including atypical forms), Flexner bacillus and enterococcus (streptococcus), were found in the stools. In other outbreaks *Bacillus mucosus capsulatus*, non-hæmolytic streptococci, *Bacillus dispar* and a monilium were identified. Virus studies have thrown no light on the identity of the causative agent. Best asks whether an organism or a variety of organisms which are non-pathogenic to older persons may become pathogenic when they are introduced into the intestinal tract of the new-born. He comments on the surprising paucity of pathological changes in this condition, which is attended with so severe an intestinal toxæmia. There is some congestion of the superficial vessels in the bowel and of Peyer's patches, and occasionally hæmorrhagic areas are visible, but there is no constant microscopic or macroscopic finding. In addition to bronchopneumonia and *otitis media*, parenchymatous degeneration of the liver and kidneys and congestion of the dural and meningeal vessels were sometimes revealed.

Best remarks that these institutional epidemics present a serious problem to the health officer, as they attack an age-group the mortality rate of which has shown the least fall. The reduction in the infant mortality rate during the past quarter of a century in New York City was about 61%. During the same period the decrease in deaths of babies under one month of age has been only 28%. The mortality rate of infants from one to twelve months suffering from diarrhœal conditions was 31 per thousand living a quarter of a century ago. Last year it was 3·6 per thousand, a reduction of 90%. For the same period the mortality of babies under one month ill with diarrhœa was 2·5 per thousand live births, whereas last year it was 1·9, constituting a reduction of only 24%. Actually in the past ten years an increase is shown. Best insists that if attempts to reduce infantile mortality are to meet with success, measures to control or prevent these epidemics must be instituted.

[1] *The Journal of the American Medical Association*, April 9, 1938.

Abstracts from Current Medical Literature.

RADIOLOGY.

Experimental Non-Calcification of Callus Simulating Non-Union.

A. W. HAM, F. F. TISDALL AND T. G. H. DRAKE (*Journal of Bone and Joint Surgery*, April, 1938) observe that complete bone repair is the outcome of two processes which usually concur: callus formation and calcification. They have designed experiments to separate the former from the latter, producing uncalcified callus. Although uncalcified callus did not show up in Röntgenograms (the picture being that of non-union), histological studies revealed it to be bony in character, as did the fact that it calcified with great rapidity under a changed experimental procedure. This illustrates the fact that complete repair of bone can be prevented by the failure of the second process (calcification), and that the two processes concerned in bone repair are controlled by entirely different factors. Like so many definitions in present-day use, certain of those used commonly in regard to fractures are decidedly inadequate. For instance, the terms "non-union" and "ununited fracture" are employed to indicate the absence of bony union. Hence "fibrous union" is an example of "non-union". It is a problem to decide whether a fracture which is united by a callus, the organic constituents of which are similar to bone but which are not calcified, should be said to present "non-union". To settle this question it becomes necessary to decide whether bone tissue is distinguished from other tissues by its organic constituents or by its inorganic constituents. The authors point out that other tissues can calcify, taking up exactly the same salts that bone does; but this does not make these tissues into bone. For instance, calcified cartilage is not bone. Conversely, although bone is almost always calcified, and although calcification is its most impressive feature, it is not its distinguishing one. In the last analysis bone is distinguished from other tissues by the character and arrangement of its organic constituents, and these are very similar in uncalcified and calcified bone. In this study fractures were produced in animals which were being fed on a diet that deranged their calcium and phosphorus metabolism. At the end of three weeks Röntgenograms gave the impression that no union had occurred, but histological examination revealed the presence of an excellent callus of bony tissue, although it was uncalcified. In similarly treated animals, allowed to live four days longer and given vitamin *D* during this period, a rapid calcification of the bony callus that was already in existence was observed. This rapid calcification of callus was noted in the Röntgenograms. The histological appearance of the fractures changed little in this time. Non-calcification of a callus is not non-union, because bony union is judged as such by the character and arrangement of the organic constituents of callus and not by its mineral content. The authors state that vitamin *D* and other so-called calcifying agents do not control the formation or growth of a callus, and only indirectly affect its calcification.

Spinal Extradural Cyst and Kyphosis Dorsalis Juvenilis.

RALPH B. CLOWARD AND PAUL C. BUCY (*American Journal of Roentgenology*, November, 1937) state that extradural cysts of the spinal cord which give rise to symptoms during adolescence are usually associated with deformities of the thoracic part of the spine, usually *kyphosis dorsalis juvenilis*. As a result of compression of the spinal cord the cysts give rise to severe spastic paraplegia, with slight sensory changes, and usually without pain or disturbance of the function of bowel or bladder. Antero-posterior Röntgenograms demonstrate an increase in the size of the spinal canal. The interpedicular distance at the level of the intraspinal lesion is increased. The pedicles are reduced in thickness. The medial surfaces may be flattened or concave, or the pedicles may be reduced to thin lamellæ. In the lateral view an increase in the antero-posterior diameter of the spinal canal with concavity of the dorsal surfaces of the vertebral bodies may also be seen. The earliest change in the vertebral bodies is a rounding off of the antero-superior and antero-inferior corners. The erosion of these corners continues and small step-like defects appear at these points. A reduction in radiodensity of the anterior one-half or one-third of the bodies is also noted. The superior and inferior surfaces of the bodies become irregular, and prolapses of the intervertebral disk or *nucleus pulposus* into the body occur. The intervertebral space becomes thinner and cloudy, and the bodies may even come in contact one with the other. In advanced cases the involved bodies may collapse anteriorly and assume a wedge shape. The authors suggest that these bony changes may be produced in the following way. A meningeal diverticulum (a spinal extradural cyst), which fills the epidural space, is in communication with the spinal fluid. The pressure within it will therefore be the same as that of the spinal fluid, namely, 300 millimetres of water. With straining, lifting *et cetera* the pressure will be raised to even higher levels. This pressure, since the cyst fills the epidural space, will be transmitted directly to the epidural veins, including the veins which drain the vertebral bodies, which are adapted to function at atmospheric pressure. Such an increase in external pressure will not interrupt the flow of blood in these veins, but will result in an increase in venous and capillary pressure and a consequent vascular dilatation or hyperæmia of all veins and capillaries behind this compression. Such a pressure, 300 millimetres of water, will not compress the spinal cord, and hence it will not of itself produce nervous symptoms, but will cause a hyperæmia in the vertebral body. The authors believe that venous congestion and stasis are responsible for the destruction of the vertebral bodies, and that such a vascular abnormality, in order to be effective, must occur in growing vertebræ at a time when they are subjected to considerable compressing force, that is, during adolescence. They suggest that *kyphosis dorsalis juvenilis* unassociated with such cysts may be due to venous congestion and stasis within the vertebral bodies, of unknown ætiology.

Röntgenological Aspects of Mastoiditis.

RAPHAEL SCHILLINGER (*American Journal of Roentgenology*, February, 1938) states that in fully developed mastoids the cardinal Röntgenographic signs of mastoiditis are: First, clouding or loss of aeration. This sign is produced by hyperæmia, œdema, exudation, granulations or suppuration. The second sign is fuzziness or loss of sharpness of outline of the cell walls. This sign is due to thickening of the mucoperiosteum and to decalcification caused by hyperæmia. The liberated calcium is retained in close proximity to the cell wall, which is undergoing dissolution and thus produces a shadow which is broader but less dense. In this sense decalcification does not mean destruction. The third sign is coalescence or destruction of cell walls. This sign is produced by pressure necrosis resulting in the merging of several cells into one. The fourth sign is high visibility of the horizontal semicircular canal, which lies in the floor of the antrum. This sign results from decalcification of bone in the periantral triangle, which makes that area comparatively translucent. The fifth sign is high visibility of the sinus groove in the air-filled mastoid. Mastoiditis produces diminished visibility of the sinus groove in the infant's mastoid. In the infant's mastoid under normal conditions the sinus is visible because there are no cells overlying its course. With subsequent aeration the course of the sinus becomes obscured gradually, so that in the fully developed mastoid the sinus frequently is barely discernible or not at all visible. In acute mastoiditis in the infant, hyperæmia and infiltration of the periosteum overlying the sinus groove tend to obscure that groove; but in acute mastoiditis in the fully developed bone and in the transitional types, decalcification

tends to increase the visibility of the otherwise obscure sinus groove. The Röntgenogram of a large pneumatic mastoid frequently fails to show the degree of pathological change found at the operating table. Signs and symptoms of acute mastoiditis may be present before coalescence is demonstrable by X rays. In such mastoids the total cell system has a large capacity, free communication exists between the cells and considerable time elapses before the pus is pent up under sufficient pressure to produce coalescence. In the infant mastoid, interpretation of skiagrams rests upon the presence or absence of aeration and the sharpness of cell outlines, especially of the cells in the angle formed by the line of the tegmen and the sinus groove. In some cases loss of cell outline is of greater significance than the apparent presence of aeration. X ray findings in mastoiditis of infants are frequently inconclusive.

PHYSICAL THERAPY.

A Dosage System for Interstitial Radium Therapy.

RALSON PATERSON AND H. M. PARKER (*The British Journal of Radiology*, April and May, 1938), working in the Holt Radium Institute, Manchester, contribute a further article on their dosage system for γ ray therapy. The actual evolution of interstitial dosage measurement in this clinic was such that two quite separate types of implantation were developed, namely, planar implants and volume implants. As described in their previous article, the authors' dosage system consists of dosage graphs and distribution rules used in conjunction; and these are given for the two types of implant. There are very few situations in which it is not possible to devise a potentially satisfactory tissue implantation with one or other of these types of implant. Experience has shown that both are of real clinical value. The planar type of implantation has been in use at the Manchester clinic for over six years; and the volume dosage method has had over two years' clinical trial. The question of tissue tolerance to interstitial implantation is discussed and the safe dosages, calculated in international *r* on this system for different types of implantation in various situations, are given. Practical applications of the system, showing the use of the rules and graphs and the mathematical calculations, are described in detail. The second part of the article deals at length with the physical foundations on which the whole system is built.

Röntgen Therapy of Dupuytren's Contracture.

S. R. BEATTY (*Radiology*, May, 1938) states that surgical treatment of Dupuytren's contracture in the hands of a competent surgeon may be excellent, but that the chief objections to this method of treatment are the danger of infection, the possibility of injury to the small but important vessels and nerves, the prolonged period of immobilization in extension required, and the disinclination of many to submit to a rather serious operative procedure. A method with none of these disadvantages, which can be carried out without loss of time or the causing of pain or inconvenience to the patient, should, if the results are at all comparable, be preferable to surgery. The author describes the results obtained by treating by X rays ten patients suffering from this condition. The technique was based on the administration of moderate doses (100 to 200 *r*, measured in air) of lightly filtered X rays at intervals of a week for three to four weeks. In seven of the ten cases improvement occurred, with loss of pain, reduction of palmar nodes and relief of contracture. In one or two advanced cases the relief was pronounced. Because of the absence of risk of X ray therapy in skilled hands, the absence of pain, avoidance of admission to hospital and interference with occupation, the author believes that this method of treatment should be considered before the more radical surgical procedure is advised.

Tissue Dosage and Radiation Effect in Operable Cancer of the Breast.

F. E. ADAIR, E. L. FRAZELL AND E. H. QUIMBY (*Radiology*, May, 1938) present a study of a series of 201 patients with primary operable mammary cancer who were subjected to preoperative irradiation in varying amounts. One hundred and thirty-eight patients were treated by 200 kilovolt X rays, and 63 patients by the four-gramme radium element pack. Approximately eight to ten weeks after completion of the preoperative irradiation, radical mastectomy was performed. The tissues were then subjected to meticulous histological examination. In every case an aspiration biopsy showed the presence of carcinoma before the irradiation was started. Also careful measurements were made relating to the exact size of the tumour, its thickness, depth and position in the breast. The doses delivered to the tumours were calculated in terms of threshold dose. In the radium bomb series the primary lesion was usually treated first, two ports, an anterior port and a lateral port, being used. Following this, three axillary ports were treated. In the X ray series the treatment of the primary lesion and of the axilla was carried out in a single course, two ports being treated daily, with 300 *r* each (measured in air), to a total of from 1,200 to 2,400 *r* per port. Tissue doses were calculated from "isodose" curves made with a small air-wall ionization chamber and water phantom. For the X rays in this series the threshold dose is about 500 *r*. With all the physical factors of radiation treatment at hand, a study was then made correlating the tumour dose with the resultant radiation effect in the tumour. Differing radiation effects were grouped according to a scale of microscopic changes. In many cases the effect of radiation on the tumour was rated as zero, even though a large tumour had clinically disappeared under therapy. The pathologist reasoned that a case in which any unaltered cancer cells were found at operation, even though 99% of the tumour had clinically disappeared, should be listed as "radiation effect zero". The authors have drawn the following conclusions: (i) The clinical impression as to the residual tumour can be fairly well relied on to express the radiation effect obtained. (ii) The larger the minimum tissue dose delivered to the tumour, the more likely is a profound radiation effect to be obtained. (iii) It is necessary to deliver by X rays at least six threshold skin doses within three weeks, and with radium almost three threshold skin doses within six days to the deepest portion of the tumour, if the maximum radiation effects are to be obtained. (iv) There is a definite relationship between the size of the tumour mass and the radiation effect obtained. The chance of obtaining a high radiation effect decreases as the size of the tumour increases. Since tumours vary enormously in size as well as in the amount of overlying tissue, it is evident that from the same beam of irradiation very different doses may be delivered to the most deep-lying tumour cells. The inadequacy of expressing radiation dosage as a certain number of "Röntgens" or of milligramme-hours per port is evident. The authors point out that no really valuable information regarding the necessary dose of radiation will be available until the actual doses delivered to the tissues in question have been measured in a large series of cases and correlated with the observed effects.

Evolution of Radiotherapy in the Treatment of Certain Generalized Affections.

R. GILBERT (*Radiology*, February, 1938) discusses the modern methods of radiotherapy in chronic leuchæmia, essential erythrocythæmia and malignant granulomatosis, with special reference to irradiation of the entire body. He issues a warning against the practice of repeating X ray therapy too frequently in leuchæmia, as it is important to defer the appearance of radioresistance for as long as possible. In erythrocythæmia irradiation of the bone marrow has yielded brilliant results, but is not always successful. In these cases, irradiation of the entire body has given encouraging results. In Hodgkin's disease the author is opposed to irradiation of the whole body, and prefers the usual method of local X ray therapy.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 23, 1938, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. B. T. EDYE, the President, in the chair.

Diverticulitis.

SIR CHARLES BLACKBURN read a paper entitled "Diverticulosis and Diverticulitis" (see page 405).

DR. H. R. SEAR read a paper entitled "Diverticulosis and Diverticulitis from the Radiological Point of View" (see page 409).

DR. J. C. STOREY said that no one medical man in a lifetime in Sydney gained enough experience to be an authority on diverticulitis. At the Royal Prince Alfred Hospital 130 cases had occurred in twenty-eight years. This meant that each member of the honorary staff got 0·4 patient *per annum* perhaps. He could talk only on impressions, which were very dangerous. Dr. Storey urged the importance of avoiding operation on persons suffering from diverticulitis. Diverticulitis was very rare and very interesting. Every little while a patient was encountered suffering from some condition in the lower part of the abdomen. Certain surgical emergencies arose as the result of diverticulitis. Most diverticula were probably congenital or there was a tendency to their formation from birth. Most people who had diverticula suffered not at all. The complications that might arise and necessitate urgent surgical treatment were: first, a very acute condition of diverticulitis with surrounding inflammation, which amounted to a physiological putting out of action of that segment of the bowel. In such a condition, what was to be done? To what was the condition due? The answer to the second question did not much matter; the surgeon's duty was to relieve the obstruction as quickly and as safely as possible. Dr. Storey advised the performance of right lumbar colostomy. This operation had fallen into disrepute; but it had been used by MacCormick, Paul and others. The details of the operation were described in Binney's book on operative surgery. When the bowel was distended, it was very easy to perform; it could be done under local anæsthesia, and the patient was absolutely safe. No doubt the critics would ask what would happen if the surgeon was wrong; if the surgeon had eyes and ears, he would not be wrong. Dr. Storey went on to detail the history of a patient he had encountered a few years previously who had an intestinal obstruction. The obstruction was thought to be in the small intestine. When the abdomen was opened, the obstruction was found to be in the colon and was accounted for by a congenital abnormality.

The second complication in which operation might be required was abscess, formed as a result of acute diverticulitis. Dr. Storey said that this reminded him of the danger of being too zealous in giving an enema. He remembered a man who had been taken into hospital and given an enema, and who had experienced great pain while the enema was being given. At operation pus and fæces were found free in the peritoneal cavity. What could be done with a patient who had a diverticular abscess? The answer was, as little as possible; the ideal was to drain the abscess, drain the bowel above it, and do no more.

Speaking of the patient without definite obstruction, but with some discomfort and upset of bowel function, who appeared to be threatened with obstruction, Dr. Storey said that if the tumour was a diverticulum it was more likely to be tubular; in carcinoma the tumour was more discrete and smaller. The general appearance of the patient was a help in diagnosis; people who were round and fat were much more likely to get diverticula than lean people. People in middle life were usually the victims of diverticula; and yet Dr. Flynn had had a patient, a youth, aged eighteen years, with urgent diverticulitis.

Speaking as a surgeon, Dr. Storey said that he would pass on the information given by a radiologist, that it was good to give these people iodides; he did not know why, but iodides always seemed to do some good. With regard to the treatment of patients, apart from surgery, Dr. Storey said that in Lockhart Mummery's book it was stated that the consistency of the fæces should be about that of pomade. How was this to be attained? There were objections to paraffin: the anal mucous membrane was unable to distinguish paraffin from flatus; further, if the patient had too much paraffin the intestinal villi became coated, and he was bound to grow thin. A good deal of harm could be done by the rough administration of an enema. Dr. Storey said that he wished to suggest the formation of a society for the abolition of the bone enema nozzle. No instrument should be blindly introduced more than 7·5 centimetres (three inches) into the rectum. In this connexion he spoke about a patient on whom he had performed a double herniotomy, and who had become violently ill. Dr. Storey had become very alarmed, and wondered what he could have done. Sir Alexander MacCormick, who had been called in consultation, asked what kind of enema nozzle had been used. The man had a slough in the rectal wall, due to injury by the enema nozzle and the injection of turpentine into the pelvic tissues. It was an insult to a man to have anything thrust up the rectum; the surgeon should not add injury to insult. Sigmoidoscopic examination was best made with the patient in the genu-pectoral position and without general anæsthesia. Another condition that was confused with the presence of diverticula and with carcinoma was perforation of the visceral peritoneum by a foreign body. Dr. Storey said that he had once operated on a man and had observed without opening the peritoneum what he took to be a huge carcinoma. He performed lumbar colostomy, and later operated again, to find that the tumour had disappeared and that a large snapper bone was present. In conclusion, Dr. Storey spoke of a patient who had been recently admitted to hospital dying of intestinal obstruction, and who had had a colostomy performed by Sir Herbert Maitland thirty years previously. She had lived in comfort for thirty years; and the *post mortem* examination revealed that the original disease had been diverticulitis and the cause of the recent obstruction of the small bowel had been fixation of a loop in the scar tissue.

DR. HAROLD RITCHIE said that he had greatly appreciated both papers; he thought that Sir Charles Blackburn had not laid sufficient emphasis on the existence of healed diverticulitis and of mild recurrent diverticulitis. These mild recurrences were often encountered, and the patients were best helped by a bland diet without very much roughage. With regard to the use of aperients, Dr. Ritchie said that some form of lubricant was very helpful in maintaining the normality of the colon. The best type was some form of agar-agar or paraffin. Dr. Ritchie was disappointed to hear that Dr. Storey thought, because not many cases of diverticulitis had been encountered at the Royal Prince Alfred Hospital, that not many cases occurred. Major cases were infrequent, and the patients admitted to public hospitals were suffering from the major complications of diverticulitis. Quite a number of patients with diverticulitis never reached hospital because perhaps the condition never went on to abscess formation. He thought there was no doubt that in many cases the old diagnosis of *pericolitis sinistra* should have been diverticulitis of the sigmoid region. Dr. Storey had said that iodides were valuable; Dr. Ritchie had never used them. He hoped that it was true that they were of value. Treatment offered a great task for the ingenuity of the medical attendant.

DR. J. G. EDWARDS, speaking of the frequency of the condition, said that almost 100 cases passed through the X ray department of Sydney Hospital in a year, but the patients were not admitted to hospital unless some severe complication was present. A Higginson syringe should

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

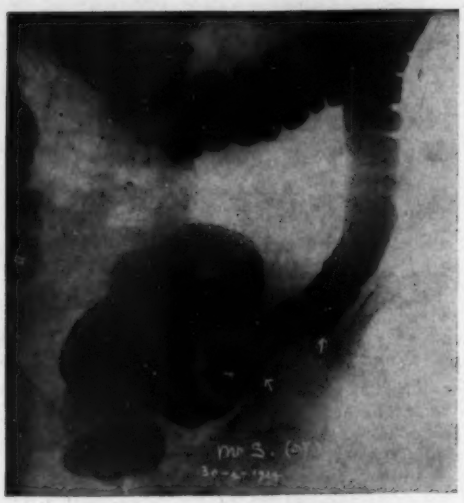


FIGURE I.
A skiagram illustrating the prediverticular state.

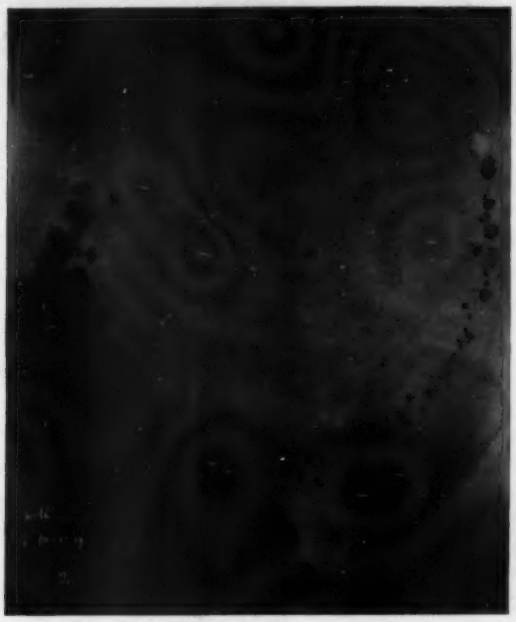


FIGURE II.
Multiple diverticula of the colon revealed by examination after an opaque meal examination.

FIGURE III.
A few isolated diverticula in the sigmoid region, some partly filled with transparent fæcal material.

FIGURE IV.
Scattered diverticula in the sigmoid region with diverticulitis. At operation diverticulitis and adhesions were found. This is the type of deformity that may be difficult to distinguish from carcinoma.

ILLUSTRATIONS TO THE ARTICLE BY Dr. H. R. SEAR.

Figure V.

Numerous diverticula and advanced diverticulitis causing obstruction to the barium enema. The diverticula in the descending colon contained opaque material from an opaque meal given forty-eight hours before.

Figure VI.

A ring carcinoma of the sigmoid colon. The diagnosis in this case is obvious from the type of defect.

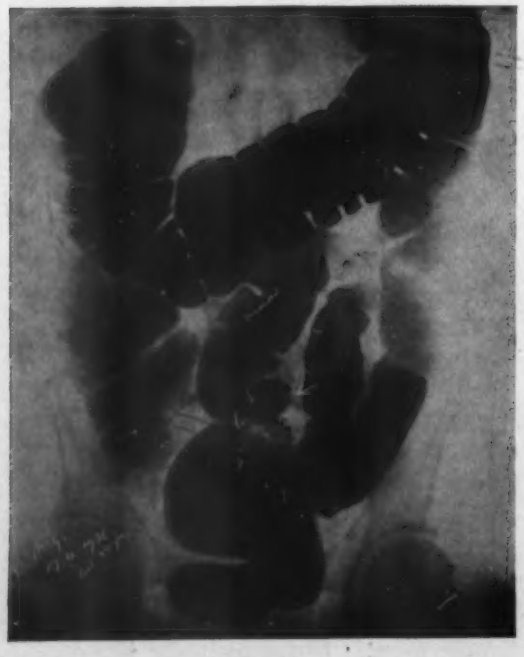


Figure VII.

Carcinoma of the sigmoid colon.

Figure VIII.

Carcinoma of the sigmoid colon. A carcinoma in which the differential diagnosis from diverticulitis is extremely difficult. Compare with Figure IV.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE IX.

Congenital abnormality of the bowel, the small bowel lying to the right, the large bowel to the left, the caecum being in the true pelvis. This abnormality is revealed by approximately one in every two thousand opaque meals. Appendicitis occurring in an appendix in this situation might easily lead to the diagnosis of diverticulitis.

FIGURE X.

Adhesions supervening on an abdominal section.

FIGURE XI.

Adhesions after a section, leading to obstruction. This film was taken after evacuation of the bowel below the site of obstruction.

FIGURE XII.

Pedunculated polypus in the descending colon. The X ray diagnosis was polypus. At operation a pedunculated polypus was removed.

ILLUSTRATIONS TO THE ARTICLE BY FRANK J. FENNER.

FIGURE III.

Section of juxtaarticular nodule in Case I. The vessels and round cells were situated in the outer looser fibrous tissue; the greater part of the nodule consisted of denser relatively acellular tissue, as shown in the lower part of this section. (× 80.)

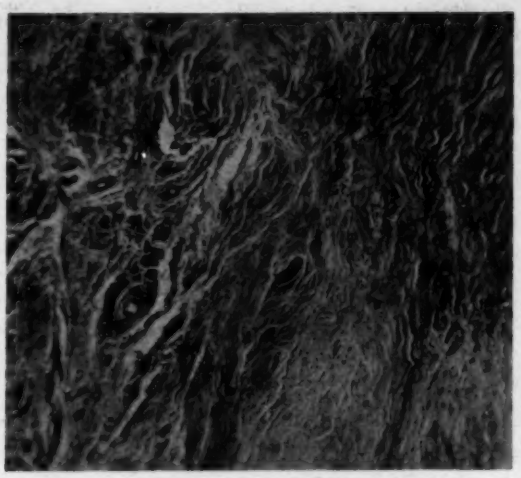


FIGURE IV.

Section of juxtaarticular nodule in Case II. This section shows the dense fibrous tissue. It was more cellular than in the previous case, with large accumulations of round cells in the neighbourhood of the vessels. (× 80.)

ILLUSTRATIONS TO THE ARTICLE BY A. R. TREMAIN.

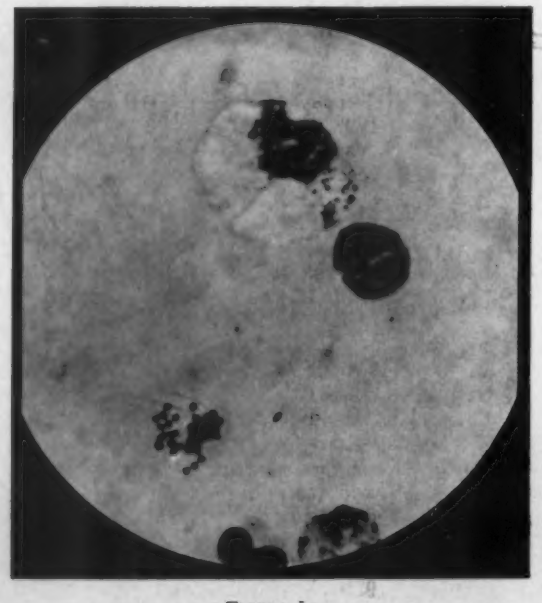


FIGURE I.

Smear of the pericardial fluid of mouse *H*, showing intracellular and extracellular L.C.L. bodies, of sizes varying from the elementary (0·2μ to 0·4μ) to the large forms (about 1μ). (Photomicrograph taken by Mr. F. H. Taylor.)

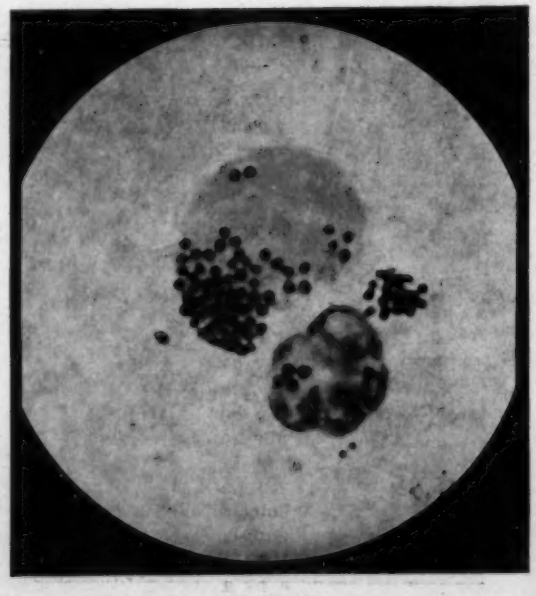
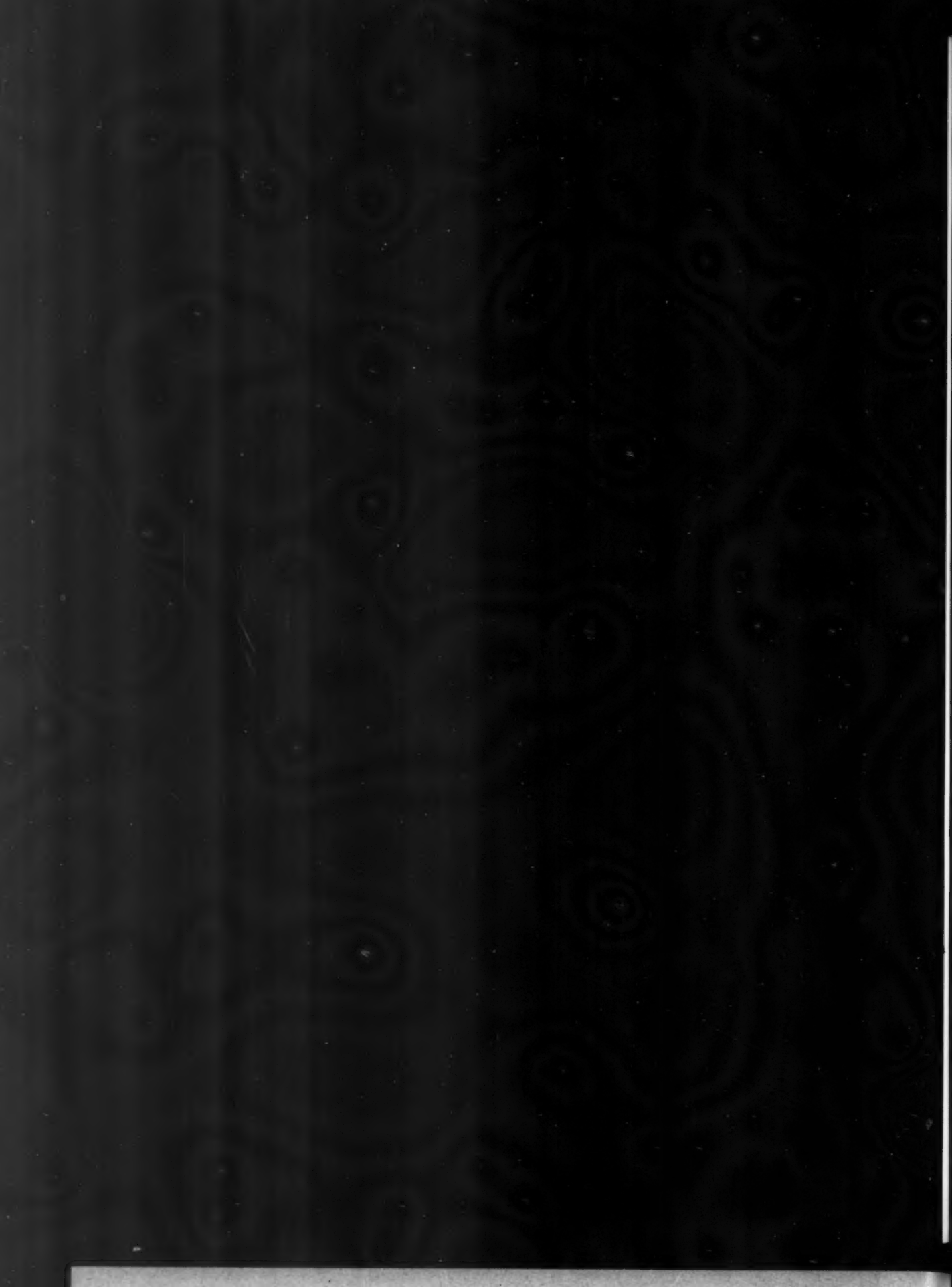


FIGURE II.

Smear of the pericardial fluid of mouse *H*, showing intracellular large forms of the L.C.L. bodies. (Photomicrograph taken by Mr. F. H. Taylor.)



never be used for enemas. Fluid from a can at a height of 45 to 60 centimetres (eighteen inches to two feet) above the patient would readily flow round to the caecum. If difficulty occurred on account of spasm, the can could be lowered and the fluid allowed to flow back. By this means the patient was rested. The can could be raised again later, when the spasm had passed. If this was done, the enema could be given successfully.

DR. GEORGE BARRON said that he had been very interested by the papers and discussion, because he had had considerable experience of diverticulitis; he had been afflicted with it himself. He had had a number of attacks, some years previously, resembling influenza, until the condition was diagnosed radiographically. He had also encountered five or six other patients during the last few years with similar symptoms. Treatment was by the use of paraffin oil always and by weekly irrigation of the bowel with warm saline solution, the can to be no higher than 45 centimetres (eighteen inches). Everything was removed from the diverticula by irrigation. If irrigation was neglected, pain and discomfort returned. The diverticula in his case were on the left side, and gave the impression that something was wrong with the bladder, as discomfort was relieved after emptying of the bladder. The correct diagnosis had been made about seven or eight years previously. Dr. Barron had not used Dr. Storey's iodide treatment, and was sure it would not be necessary. People seldom died of diverticulitis, but tended to live on to a ripe old age.

DR. CLARENCE READ said that he had been very interested also, because he too had suffered from diverticulitis for a good many years, and had had two very acute attacks, with no very serious complications. He had found difficulty in the fact that he could not have a purgative and could not take paraffin. He had solved the problem by means of fruit—apples in the winter and plums and such fruits in the summer—and by keeping the lower part of the bowel empty. This he accomplished by means of 115 cubic centimetres (four ounces) of warm water or warm olive oil, which were left in all night. He also endeavoured to have a bowel evacuation twice a day. Tincture of belladonna relieved the spasm which often caused great pain in acute attacks.

DR. R. A. MONEY said that he thought that diverticulitis was relatively common. Recurrent mild attacks were met with often in the surgical outdoor department. Complications were perforation leading to general peritonitis, or a local abscess, which might rupture into the bladder. When rupture into the bladder occurred the patient suffered from that distressing complication known as pneumaturia. Dr. Money stressed the need for care in the diagnosis of recurrent attacks once it had been found that a patient was subject to diverticulitis. It was important that the diverticulitis should not be blamed for every kind of abdominal pain. Another pathological condition could quite easily be the cause. Dr. Money mentioned a man who had been regarded as suffering from recurrent attacks of diverticulitis, and who had become more and more ill. Actually, he had had an appendiceal abscess that had finally ruptured into the bowel, with spontaneous cure. With regard to treatment, Dr. Money said that colonic lavage with saline solution at a temperature of 40·5° C. (105° F.) was very valuable in the acute stages, and should be continued once a week for the rest of the patient's lifetime. It was not very inconvenient, and could easily be carried out by the patient himself. Something in the form of an agar-agar mixture was infinitely better than paraffin as an aperient.

Sir Charles Blackburn, in reply to Dr. Ritchie, said that he thought he had laid sufficient stress on recurrent cases of diverticulitis. Experience seemed to show that about 60% of patients with diverticulitis recovered and apparently had no further symptoms. About 40% had further trouble. He thought that the 60% got on quite well without having to carry out any special treatment. He knew several people who had had severe attacks and who carried out no treatment beyond ensuring regular evacuation of the bowels. Sir Charles referred to the unfortunate results of too much solicitude on the part of the medical practitioner. One man had had rules laid down for him and stated that he had a decent meal only when he went to his club, because his wife insisted so strictly on adherence to his diet. Even if the very greatest care was taken, there was no guarantee that these people would not suffer other attacks. One thing he wanted to stress was that a patient should not be told that he had diverticulitis when he had not.

Proceedings of the Royal Commission Appointed to Inquire into Matters Pertaining to National Health Insurance.

Wednesday, August 31, 1938.

THE examination of Dr. T. H. Small *in camera* was concluded, and he was examined and cross-examined in open court. Dr. Small gave evidence concerning his relationship to certain public hospitals. Bad debts were then discussed.

Mr. Bowie Wilson: In your income tax figures you have an amount of £102 for drugs. Can you give us an idea as to what proportion of that would be used for lodge patients as against your private patients?

A.: A very small proportion—a negligible proportion—would be used in the case of lodge patients. A lodge patient who requires the use of drugs, dressings, sticking-plaster and the like is almost always a workers' compensation case.

Q.: The bulk of that £102 would be for anæsthetics?

A.: A certain amount, but not the bulk, would be for anæsthetics.

Q.: For use on private patients, in the case of confinements and extra lodge work?

A.: In the case of confinements there would be only a small quantity of ether. About £3 worth of ether would last me about twelve months.

Q.: Under this scheme you will be allowed to send in a bill to the department for any drugs you use on your patients. You say that that will be of no advantage to you because the amount is negligible and not worth considering?

A.: What I would actually use I do not think would be worth consideration.

The Chairman: Why should the proportion of drugs and appliances be considerably less in respect of lodge patients than in respect of private patients?

A.: Take the case of whooping-cough. The vaccine for that is fairly expensive. The vast majority of lodge patients would send to public hospitals any of their children who had whooping-cough. In the case of diphtheria preventive, a dose for a contact might come to about fourteen shillings' worth of antitoxin.

Q.: A serum of that kind—is not that covered by the lodge?

A.: No, they buy that themselves at their own dispensary.

After examination concerning the midwifery branch of his practice Dr. Small was examined by Mr. Williams. Extracts from this evidence appear below.

Mr. Williams: For the purpose of arriving at your percentage of loss due to national insurance you made a count of heads?

A.: Yes, for one month.

Q.: On a count of heads dealing with persons to whom included services were rendered you arrived at a figure of 262 out of 853, or 31%?

A.: That is so.

Q.: So that out of 853 you estimate that 262 would come under national insurance?

A.: Yes.

Q.: Do you know how many of those were lodge patients and how many private patients?

A.: None of them were lodge patients.

Q.: So that the figure is not subject to any reduction because of the high rate of lodge patients who will go into national insurance?

A.: It will not be subject to any reduction.

Q.: Assuming that you had a class of patients, lodge patients for instance, and that they took up considerably less time than the private patients, it would be reasonable to charge them less?

A.: Assuming that to be true, yes; but a lodge confinement does not take any less time than a private one.

Q.: But you get paid for lodge confinements?

A.: I get paid less for them.

Q.: Whatever the position, they are included among your private patients for the purpose of these calculations?

A.: For certain calculations only.

Q.: As a matter of fact, according to your own figures lodge patients are charged less. The return from them is 3·4 as against 7·8 for private patients?

A.: The figure of 7·8 has been altered. I cannot admit that I give lodge patients only half the time that has been given to others.

Q.: I gather from your evidence that you have a very fair knowledge of your lodge patients?

A.: Yes.

Q.: May we take it that it is only an exceptional lodge patient who imposes on you?

A.: They do not actually impose on me; but sometimes they do not think. There are impositions, but I do not think that they are deliberate. Sometimes, for instance, a woman might have a lot of children to look after, and she may not have a high order of intelligence; but one meets the same kind of thing outside lodge patients.

Q.: But the person who is paying your ordinary private fee has not the same consideration for you as the lodge patient has?

A.: Certain classes of people simply do not think; for instance, in the making of night calls. I actually do more night calls for persons not under capitation fee than for lodge patients.

Q.: The lodge patient is bound by contract which has certain terms in it?

A.: Yes, but they are not always observed.

Q.: By either side?

A.: I do not always bind them to the contract.

Q.: But you agree that because a charge is made for a night call there is less tendency for lodge patients to make such calls?

A.: That charge is not always enforced. It is stipulated that the calls must be made before a certain time; but we do not always bind them to that.

Q.: You do not suggest that the conditions in the agreement make no difference in regard to the number of night calls by lodge patients?

A.: I do not think it actually makes a great deal of difference.

Q.: I think you said yesterday that as a deterrent to the making of unnecessary night calls when a person first came to your area you charged him for such calls on the first occasion in order to discourage him?

A.: Not always. I make such a charge only when they could have sent for me during the day and did not do so until after 8 p.m. It is just to let them know that I am entitled to make the charge. As a matter of fact, for the year 1936-1937 I charged for only two night calls to lodge members and made such charges in respect to three lodge members' dependants, that is, five in all.

Q.: Do you know how many night services you gave?

A.: I do not know; but it was a lot—it was in fact an irritating number. As a matter of fact, one gets a large number of calls at night that could very well have come in during the day.

Q.: I understood that it was for that kind of call that you made the charge?

A.: Not always. In my particular case I am engaged on other work during part of the day and have to attend partly to my practice at night.

Q.: Another condition of the contract is that whenever a lodge patient is physically able to do so he must attend your surgery?

A.: Yes.

Q.: And I take it that most of the lodge patients do so?

A.: Yes, but very often I have been called out to people who were physically able to attend.

Q.: But that condition in the agreement must have the effect of bringing along to your surgery a big proportion of lodge patients?

A.: Yes.

Q.: And that result does not apply to private patients?

(No answer.)

Q.: Is it not a fact that a private patient who happens to be unwell will not hesitate even at 7 or 8 o'clock at night to ring for a doctor?

A.: Not necessarily. I do not think there is much difference in the relative demands made in that way.

Q.: So that you do not think that the clause about attending the surgery if possible has any effect?

A.: I do not think it has much effect.

Q.: Many of the services that you render to friendly society members or patients are what you term repeats?

A.: Yes. They may send the book up to the surgery with a request that it be repeated.

Q.: And that is termed a service?

A.: Yes.

Q.: Those kind of services would not require much time?

A.: If they just send the book along by a child on its way to school, saying they want a repeat, then very little time is taken; but if the patient comes in himself and opens a conversation one must listen to him and sometimes a good deal of time is taken up.

Q.: We have been told the lodge patient is more inclined to go to the doctor than is the private patient, because the private patient must pay a fee. Therefore, when a private patient attends he is apt to take up your attention for some time?

A.: Not necessarily.

Q.: But you will agree that as a general rule private patients' service will take longer?

A.: If you include in the friendly society service the number of times patients merely send their books along I will agree.

Q.: The position is, you treat lodge patients and private patients in exactly the same way?

A.: I do. I do not think there is much difference between the time devoted to the two classes of patients.

Q.: Even though you may include in the case of lodge patients those repeats, and issuing of certificates *et cetera*?

A.: That might make some difference, but it would not be very great. Many private services are very brief. The lodge services in the mass would be smaller, because they include repeats, but where a patient comes in with his book there is very little saving of time.

Discussion later took place concerning the tender of income tax returns of the witness for a period of five years.

Mr. Bowie Wilson: For the purpose of having them on the record, I wish to have included in the *in camera* evidence of this witness his taxable incomes for the years 1932 to 1936-1937, and at the moment I merely wish to show these figures to the Commission.

Mr. Abrahams: I object to this. My friend is tendering these figures, I understand, because of a five years'

average appearing in the left hand corner of this assessment. We have had a period of depression which, I think, started in 1932-1933 and continued till 1935-1936. I do not mind going back three years, but surely some limit must be set. If I have to ask doctors what factors operated in 1932 to reduce their income to *X*, we are going a long way from reality by going back five years. The real question is, what is their income now. As Your Honour said yesterday, the best way to test what their services are worth in any particular case is to see what their income is now. It may be that illness operated in a particular year. Furthermore, there was a depression, and neither I nor the Commission, I suggest, wants to deal with the figures for the past five years. If my friend thinks that something ought to be tested because the figure for a later year seems inordinately high or low, then he can do it; but to tender that barely and force me into a situation in which I have to inquire into all the cases over a period of five years, I submit that is not proper.

The Chairman: We think it would be a reasonable thing to take the three-year average, that is, beginning with 1934-1935 and running up to 1936-1937.

Further discussion ensued on this point, and Sir George Mason Allard stated: We are concerned more particularly with the gross income before the deductions are made. The gross income is what we are interested in, because we have to determine what amount is to be paid to the doctor. What expenses he has to come off is his matter; we want to compare gross income with what we are going to give him, which is gross income.

The Chairman: That may be in so far as working expenses may vary under national insurance as compared with present conditions.

Sir George Mason Allard: His expenses will perhaps be much the same; certainly the question of drugs will not come in, because he will be paid for them separately; but other expenses, such as rent, secretary and motor car, all have to be borne and deducted from what he receives from this Commission. The income tax return is useful in that it is a confirmation of what evidence has been given of his gross income. I think we are concerned more with gross income over the period than with net income.

Dr. Small was then further examined *in camera*.

Dr. Charles Walter Coombe was examined *in camera* and later in open court. He was first examined as to what examinations were made on the entrance of new lodge members and generally as to lodge practice.

Mr. Bowie Wilson: Amongst the dependants there are children up to the age of sixteen years?

A.: Yes.

Q.: I suppose that in your district, in this class of lodge patient, some of the children between the ages of fourteen and sixteen years go to work?

A.: Yes.

Q.: And you have to attend to them under the lodge fee?

A.: Yes, up to sixteen years.

Q.: Under the national insurance scheme you will be paid for them as adults?

A.: Yes, after they are fourteen.

Q.: Provided they are in employment.

A.: Yes.

Q.: Any girl over fourteen years who goes into domestic service is treated as an adult.

A.: Yes.

Q.: So that you will have some extra payments from your lodge people under that head?

A.: Yes.

Q.: You will be getting paid for those whom you now treat for the ordinary lodge fee?

A.: Yes.

Q.: Some of those who are working two or three days a week at Lysaght's—are they lodge people?

A.: Quite a number of them are.

Q.: Do their wives go to work, doing washing and so on?

A.: Some of them do.

Q.: Under the scheme you would get paid for those?

A.: I would, but they are very few. Some of these people have families and the wife has to stay at home to do the work.

Q.: There is nothing on the cards to show what the number is?

A.: No, I could not say.

Q.: But to that extent you would get extra payments?

A.: We will get 22s. instead of 26s.

Q.: And if there were a boy or a girl going to work you would get 33s.?

A.: Yes.

Q.: Have you taken that into consideration in estimating your loss?

A.: That would be very small.

Q.: Which: the boys and girls aged fourteen to sixteen years who are working?

A.: No, the women who go out to work.

Q.: But those between fourteen and sixteen years who go to work would be large in number?

A.: They would number more than the wives who work.

Q.: What percentage of the children between fourteen and sixteen years work?

A.: I could not tell you that; I have not gone into that matter.

After examination as to bad debts, Dr. Coombe was further questioned by Mr. Bowie Wilson.

Q.: You have stated that you consider that out of 4,489 attendances you will lose 30% under national insurance?

A.: Yes.

Q.: That works out at 1,346 visits?

A.: I believe so.

Q.: How do you arrive at that figure of 1,346 attendances?

A.: I counted heads for three months, and arrived at the conclusion that I would lose from 35% to 40% of patients. That was an estimate.

Q.: Did you keep a record of the number of visits during those three months?

A.: No, I did not count the visits.

Q.: You checked all your patients for three months?

A.: Yes.

Q.: And presumably you considered which of those patients would become insurance members under national insurance?

A.: That is right.

Q.: The number of attendances for lodge patients you gave as 3·3 a year. Is it the same for private patients?

A.: I suppose so.

Q.: So that you would expect to visit your private patients 3·3 times a year each?

A.: I did not count the private patients.

Q.: When making your estimate did you take into consideration the age of the patient?

A.: I considered his age in order to determine whether he would come under the insurance scheme or not. I would not count women over sixty or men over sixty-five, because they would be outside the scheme.

The Chairman: When you excluded them from the list of possible insurance patients, that meant that you put them amongst those who would be left as private patients. You would not exclude them altogether?

A.: They would be still potential private patients.

Mr. Wilson: You gave a figure of 2·6 as representing dependants of lodge patients?

A.: Yes.

Q.: Did you simply make a record of the members' names and then add on a number for dependants representing 2·6 for each member?

A.: I do not see how the figure of 2·6 comes into it.

Q.: Some of the patients will be children?

A.: Yes.

Q.: You will have to look back and see what the child's father is, and determine whether he is likely to come under national insurance or not?

A.: That is so.

Q.: Did you do that with each individual visit, or did you merely look at a lodge list and make up your mind who would be going out?

A.: I went through the attendance book and considered the individual names.

Q.: Can you give us any assistance in finding out what percentage of your 4,489 services to private patients were in respect of persons receiving less than £365 a year income, which would bring them under the *Insurance Act*?

A.: I think I answered that before. I said that, on going through my books for three months, I reached the conclusion that I would lose from 35% to 40% of patients.

The Chairman: But you can lose them for more than one reason. First of all you can lose them because they have not enough money, and you can keep them because they are too old to come under national insurance or because they have too much money; but if their years are few enough and their incomes small enough, then you will lose them. How many will you lose because they have not enough money?

A.: I could not say how many of the 4,489 would be lost because of the smallness of their income.

Mr. Wilson: I also had in mind the fact that such persons as small contractors and shopkeepers, and other self-employed persons, would not come under national insurance because they are not employees, though their incomes might be below £365 a year.

The Chairman: Such persons will remain as they are now.

Mr. Wilson: I was trying to find out how many of those were receiving less than £365 a year, because the man whose income is below that amount would not have too much surplus for paying 10s. 6d. a visit to a doctor.

The Chairman: That may be, but apparently they will remain in the same group as now, and as far as they are concerned the medical profession will be just as well off or just as badly off under national insurance as they are at the present time.

Mr. Wilson: It has been suggested that under national insurance there will be a tendency for very many more people to go into hospital than is the case now. Are you suggesting that a person under national insurance will be more readily able to gain admittance to a public hospital than was the patient about whom you told us this morning?

A.: I do not think they will be able to get in any more readily. It does not matter who you are; if there are no beds patients cannot be admitted, unless the Government will have some pull that others have not.

Q.: But if there are no beds they cannot get in?

A.: Presumably.

Dr. Coombe was further examined *in camera*.

Dr. Walter Terence Joseph Harris was examined *in camera* and later in open court. He was questioned concerning his friendly society practice.

Mr. Abrahams: Do you charge a night fee?

A.: Yes.

Q.: Did you always charge it?

A.: No; as regards lodge patients I used not to charge.

Q.: What did you find when you did not charge it?

A.: I had very little sleep.

Q.: Then you began to charge it?

A.: Yes.

Q.: Now you charge it?

A.: Yes.

Q.: And you find you do not have night calls?

A.: No, it was almost unbelievable.

Q.: Now you are only called out when it is necessary?

A.: Yes.

Q.: Do you think that has made any difference to the health of your patients?

A.: Not at all; if a thing is serious enough they will call you out.

Q.: Do you receive payment for services outside the lodge agreement?

A.: Yes.

Q.: Are such services increasing or decreasing?

A.: Decreasing.

Q.: Why?

A.: People are going more to public hospitals for their extras.

Q.: Is that your experience?

A.: Yes. I might add that extras that used to go in private hospitals are also decreasing as regards private practice. I think that more people who previously used to go to private hospitals are now going into public hospitals, because there has been a lot of talk by Mr. FitzSimons, the Minister for Health, who has rather rated our private hospitals, and I think people are getting a bit scared of private hospitals.

Q.: You heard Dr. Coombe express opinions highly favourable to lodge patients: that they did not summon the doctor or go to the doctor unnecessarily. Does your opinion accord with that?

A.: No. I think that lodge patients come along quite a lot. If I might compare it, lodge patients come to you for things which a private patient would not dream of coming for; he consults you for things a private patient would not go to a doctor for—minor things; he worries about them.

The Chairman: And a private patient will go to a druggist and say: "Give me something for this"?

A.: Yes, whereas the lodge patient will come up to be treated for it.

Mr. Abrahams: What is your opinion about the adequacy of the capitation fee of 26s. for the service rendered to lodge patients?

A.: I think that it is quite inadequate unless you regard the whole thing as a charitable business.

Q.: Not pure charity?

A.: No; it is a reduced fee due to the fact that they are not able to afford private fees, and also because they help a medico when he is starting.

Q.: Could you do that throughout your whole practice?

A.: No.

Q.: It is your private practice that enables you to do it?

A.: Yes.

Q.: I do not know whether you have any mileage?

A.: Very little.

Q.: Have you any views about the mileage clause in the agreement: whether it acts as a deterrent?

A.: I think any extra fee acts as a deterrent.

Q.: What is your opinion as to whether it is necessary to give more services per person to dependants than to lodge members?

A.: I think that the dependants need more per person.

Q.: Largely more?

A.: Not very largely. In one of my counts of heads for three months, out of 328 persons there were 78 members and 250 dependants.

Q.: That is services?

A.: Yes.

Q.: I was trying to get at the services per member?

A.: Do my dependants have more services than members?

Q.: Per person; do you think, by and large, a man requires more or less attention than his wife?

A.: I think the wife needs more than the man.

Q.: Largely more?

A.: It varies; when they are young the men will probably require more on account of accidents; that is counteracted to a large extent by midwifery; but when they get older the women generally require more than the men.

Q.: When the woman gets past the change of life, say over fifty years?

A.: It would be about the same then for men and women.

Q.: When national health insurance comes in, it will set free among the class of private patients two-thirds of the dependants who come under the contract practice; they will then be available for private practice, whereas they are now treated under contract. Do you think you will ever be able to get more than the contract rate or the national health insurance rate from those?

A.: No.

Q.: Do you think you will get that rate from them?

A.: No, because they will tend to become panel-minded.

Q.: It is not a matter of a new tendency; they have been contract-minded for some years?

A.: Yes.

Mr. Abrahams then questioned the witness as to which paid the better: the included service, which he described roughly as a physician service with minor operations, or the excluded service, which he defined as rather a surgical service.

Q.: Take both sides of the picture and see whether you regard the excluded services as valuable per service as the physicians' service? I know that there are some physician services such as difficult cases which may be long, but on the other hand, some are short. What is your view on the subject?

A.: They about balance up.

The Commission adjourned at 4.30 p.m. until 10.30 a.m. on Thursday, September 1, 1938.

Thursday, September 1, 1938.

Dr. W. T. J. Harris was further examined by Mr. Abrahams and cross-examined by Mr. Dovey. His evidence was concluded *in camera*.

Dr. Noel Simon Solomons, Mittagong, was examined *in camera* and then in open court by Mr. Gain. He said that he charged a night fee to his lodge patients and he was of the opinion that it reduced the number of night calls. He had found that the return for extras from lodge patients was gradually diminishing owing to the increasing numbers of patients who went into the public wards of hospitals. His experience was that lodge patients tended to consult him more than private patients and in regard to trivial matters. He enforced the mileage fee and was of the opinion that the existing lodge capitation fee was inadequate. He gave in twelve months 250 services to 60 members and 333 services to 98 dependants, so that he gave approximately equal attention to members and dependants. He attended on an average 225 boys at the Mittagong Farm Home. In a year he gave 3,544 services for a remuneration of approximately £400. By reason of the organization and the matron's supervision he was able to see many boys in a short time.

In cross-examination by Mr. Dovey, he said that lodge patients consulted him about more trivial complaints than private patients. "The private patient will go to a chemist and get a bottle of 'Heenzo' for a cold, whereas a lodge patient will go to the doctor because he can get the medicine for nothing." Questioned as to bad debts, Dr. Solomons stated that he wrote off about 8% in a year for services rendered to private patients and not paid for.

Discussion then ensued as to lodge patients' going into hospitals and the adequacy of remuneration for friendly society practice.

The Chairman: I think you may take it that so far as I am concerned I shall not bother myself much with the question as to whether the remuneration for this service rendered to lodge patients in the circumstances that have prevailed is inadequate or not. Generally speaking, it is the price that has been accepted by the medical men for doing that work in those circumstances. It does not follow at all that they must be held to be limited to that amount in the sort of practice they will have to do under the national health insurance. But as to the work that they have done in the past, they have done it at that price. That is the price they take, and, broadly speaking, have taken for a number of years, and I am not going to concern myself with the question as to whether it has been inadequate in the past or not.

Mr. Dovey: I am obliged to Your Honour for that.

The Chairman: That is my view; I do not know whether it is the view held by my colleagues. It is just as well to express it so as to indicate the issue with which we have to concern ourselves. We have not got to decide whether the lodges treated their doctors in a niggardly way in the past; the doctors have taken it.

Mr. Williams: It may be relevant in respect of those terms of reference which have to do with the dependent services.

The Chairman: Assuming that the amount of work and the nature of the work to be done by the doctors in respect of lodge members is substantially the same as in the past, and assuming that the amount of work they will do in the future in respect of a lodge patient's dependants will be done in similar circumstances to those which have existed in the past, then the amount which is now being paid will be some guide as to what should be paid to them in future in connexion with those dependants. We have got to determine that as one of the outside matters. So that it may be we shall have to pay attention to what has been paid in the past as a guide to what should be paid in the future in respect of the dependants. In that way that may become of importance. There again we give attention to it, and we presumptively would feel bound to act upon it if the circumstances generally were the same as they have been in the past. We have got to decide whether they will be the same; but if they will be the same, then the past price is a fair guide as to what the future price will be, assuming that the circumstances will be the same.

The Chairman: Assume that the scope of the service in the future will be the same as in the past, and assume that the other accompanying circumstances will be substantially the same in the future as in the past, there then will be a pretty strong case to say that the rate for these dependants shall be substantially the same in the future as in the past; but the circumstances may change a good deal. That is one of the points. It is said by the doctors, and with good reason, that this lodge price has been a concessional price. Standing by itself without any more it would be inadequate; but we were prepared to do the work at that concessional price having regard to the extent of our private practice and other accompanying circumstances; it was worth our while to work at a concession, but in future, under the national health insurance, it may not be worth our while. And they say it will not be worth their while, and that is a matter we shall have to consider.

Mr. Williams: Assume for a moment that an examination of the facts shows that it has not been a concession in all the circumstances.

The Chairman: That, I think myself, should certainly be taken into consideration. This is the point: if it does turn out, speaking by and large, that for the work the doctors do in respect of lodge patients they receive substantially an equivalent in value to that which they receive for work done for private patients, then there is no concession.

Mr. Abrahams: This morning Your Honour asked for figures dealing with hospitals and population. I take the following figures from the Hospital Commission's report. For the year 1911 in-patients were 56,564 and for 1936, 199,484, or a percentage increase of 252·6%. Population had gone up in that time by 63%. Out-patients were shown at 116,346 in 1911, and 563,918 in 1936, or a percentage increase of 384·7%. The population in 1910, the census year, was 1,643,855, and in 1936, 2,681,736, or a percentage increase of 63·1.

The Chairman: It certainly looks as if the hospital population has outstripped the other population.

Mr. Williams: You said that the amount which you received from lodge patients is inadequate. Is that your opinion of the position?

A.: Yes. The amount received per service is much less in the case of a friendly society patient than in the case of a private patient.

Q.: Is there any other reason?

A.: I have not given much thought to any other reason.

Q.: Do you know what is the difference in respect of the amount you received from your private patients and your lodge patients, per service?

Mr. Abrahams: I admit that the result is 8s. 4d. and 5s. 8d. respectively.

Mr. Williams: Did you introduce any other consideration into the matter?

A.: Well, I think you give very many more residual services in respect of your lodge patients than in respect of your private patients. That sort of thing is not taken into account. If you have a private patient in hospital

you deal with him only in hospital; you may have one or two inquiries about him; but lodge patients come up at least every week for a certificate, in addition to some of their relatives, causing a lot of extra work.

Q.: If you gave a certificate you would include it in the service?

A.: While they are in hospital, if they are given a certificate, it is not included in the number of services.

Q.: When a member is in hospital does he not merely tender to his lodge a certificate showing that he is an in-patient, and that retains him on the sick fund of his lodge until he comes out of hospital?

A.: Not in my district; they require certificates every week.

Q.: Even while he is a patient in a hospital?

A.: Yes.

Q.: Did you consider the restrictions or conditions which are included in your contract of service in relation to visitations at your surgery?

A.: Yes. They are asked to come to the surgery if they are able to do so. Private patients do the same thing in most cases; they do not send for me when it is a simple matter to come to the surgery. I do not think that private patients call me out any more than lodge patients do.

Q.: Will you draw any distinction between the time you take on a service rendered to a friendly society member and to a private patient?

A.: I give them exactly the same service.

Q.: Would you say that the time you occupy in service to a wife is any different from that rendered to a member?

A.: I think we are called upon to spend less time with the member than with the dependants.

Q.: Per service?

A.: Yes.

The Chairman: My colleagues and I are inclined to think there is not much doubt that, generally speaking, rather more time is required to be spent with the lodge doctor in dealing with the wife, and possibly with the other dependants, than with the member, perhaps not in the actual examination, but incidentally. We think it desirable to indicate that on the evidence up to now we think that that conclusion should be arrived at. If both sides think they have no hope of disturbing that conclusion, we need not trouble any more about examination along those lines.

Dr. Sidney Keith Dwyer, Gosford, was examined in open court by Mr. Abrahams. He stated that he had been in practice for eighteen years and had had English panel practice experience. He was medical officer to five friendly societies and had on his list 146 members and 286 dependants. He did not charge a night fee when he began practice, but later imposed one, as there were too many night calls. The number of night calls dropped 50% when he imposed the fee. He had now ceased making the charge.

Mr. Abrahams: What is your opinion about lodge patients' tending to go to hospital?

A.: I think that for the more expensive services they do tend to go to hospital.

Q.: Do lodge patients demand more frequent attendance than others?

A.: I think so, particularly for minor troubles.

Q.: That is partly accounted for by the fact that they have to come to you to get medicine?

A.: Yes.

Q.: What is your opinion about the concessional nature of the lodge service?

A.: I think it is a concessional service. I do not make any distinction between lodge patients and private patients.

Q.: And the figures show how remunerative or otherwise the lodge practice is?

A.: Yes.

Q.: You enforce the mileage clause in the contract?

A.: I do.

Q.: But your visits to lodge patients outside the mileage limit are practically negligible?

A.: Yes.

Q.: I understand that you have some figures indicating whether the dependants of lodge members require more attention than do the members themselves?

A.: Yes, for the year ended June 30, 1937, I saw 237 lodge patients, of whom 77 were members and 160 were dependants.

Q.: That figure of 160 must be divided by 1·9, so that you get 84 dependants as against 77 members?

A.: That is so.

Q.: The dependants saw you slightly more than the members, as 84 is to 77?

A.: Yes, for that particular year.

Q.: Your experience is that women expect to receive a prescription while men do not?

A.: That is so.

Q.: As compared with men it may be said that women like taking medicine?

A.: Yes.

Q.: What is your idea of paying travelling expenses on the basis of one service per year for everybody over the mileage limit on the panel?

A.: I do not think that it would suit my district. From an analysis of my figures it will be seen that I get very few visits outside the three-mile radius. We get 5s. a mile one way, and the patients can travel to my surgery by bus from the greatest possible distance for 7s. return. It would be cheaper for them to come in to see me than to call me out. Then, if it is impossible for them to come in, they ring me and ask if I think it is necessary to send the patient to hospital, and if I say yes, the patient is brought in by ambulance. If the Government were paying the mileage fee the attitude of patients would be: "If we go into the doctor it will cost us 7s., but it will cost us nothing to call him out."

The Chairman: Have you any suggestion to make as to how that position should be met? These visits will have to be made by the insurance doctor, and it is only fair that he should receive a proper remuneration. Have you thought out a scheme?

A.: I think we should be paid according to the actual mileage we travel.

Q.: That means that in every case separate accounts will have to be rendered by the doctor?

A.: Yes.

Q.: The mileage will have to be measured, and so on?

A.: Yes.

Mr. Abrahams: The mileage has to be measured in any case.

The Chairman: There might be a possibility that journeys would be undertaken unnecessarily if the medical man can send in a bill for every visit he makes?

A.: We would be expected to show on the cards what kind of complaint was being treated, and this would enable the district committee to impose a check.

Dr. Dwyer gave evidence as to his experience of the panel practice system in England. He stated that he had been in England in 1935 and 1936 studying dermatology and acted as *locum tenens* at Hull in a panel practice. He believed that men engaged in panel practice were overworked.

Mr. Dovey: Where was this panel practice for which you did the locum?

A.: I did a voluntary locum. This man was a personal friend of mine and I went to stay with him in Hull. I was going to stay a week, but eventually I stayed five days, and while I was there he asked me whether it would be of interest to me to do a couple of days' work for him, and that is how I assisted him for three days.

Q.: Did you make a lot of visits?

A.: No, most of the work was consultation work.

Q.: How many visits?

A.: About twenty in three days.

Q.: You started off, as I understand it, by saying that your experience was that when you got to the door the wife would say that the husband was very ill, and ask you would she ring for the ambulance. How many times would that happen?

A.: That happened seven or eight times out of the twenty visits.

Q.: So those seven or eight instances out of the twenty visits might be regarded as initial visits for that particular illness?

A.: Out of that number I only sent two to hospital after examining them.

Q.: Do you know how many patients he had on his panel?

A.: No, but I could give you the number he used to see in a day. He used to see about 20 in the morning, 20 in the afternoon and from 60 to 70 at night.

The Chairman: Where was that?

A.: In Hull.

Q.: Did you see similar numbers in consultation?

A.: Yes, pretty well. At night-time the surgery was overcrowded; but he told me himself that he never got more than 15 visits per day.

Mr. Dovey: How long would he take to attend to the patients at night?

A.: He would start at 6.45 p.m. and finish round about 9 p.m.

Q.: He would do from 60 to 70 patients unassisted—that is what he told you—in that time?

A.: Yes.

The Chairman: That is, he would take two and a quarter hours to do 60 to 70 patients?

A.: Yes.

Dr. Dwyer was further cross-examined by Mr. Dovey concerning his experience in Hull, and was later examined *in camera*.

The Commission adjourned at 4.30 p.m. until 10.30 a.m. on Friday, September 2, 1938.

Friday, September 2, 1938.

Mr. W. Slater announced his appearance on behalf of the Friendly Societies' Association of Victoria.

Dr. S. K. Dwyer was further examined as to the general conditions of lodge practice in the Gosford district.

Dr. Roy Cadell Traill, Vaucluse, was examined both *in camera* and in open court.

Dr. Rupert Sheldon, Granville, was then examined, particularly with reference to his experience as a panel practitioner in England from 1930 to 1936. He stated that he had practised at Manor Park, in the East End of London, for nine months. The usual private fees paid there were 1s. 6d. for consultation and medicine in the surgery and 2s. 6d. for a visit and medicine away from the surgery. He attended 1,900 panel patients. From there he went to Cambridge for five weeks where the usual private fees were 2s. 6d. for consultation and medicine at the surgery and 5s. for a visit and medicine away from the surgery. There were about 1,600 panel patients. From Cambridge he went to Plymouth for four and a half years, where the private fees were 2s. 6d. and 3s. 6d. respectively. There were 1,600 panel patients. The income for extras for panel patients was practically negligible.

Mr. Abrahams: From your experience in England give us your opinion of the English panel doctor?

A.: I think he is a grossly overworked man. He naturally has to rush his work to get through the numbers, particularly during the busy months of the year.

Q.: What was your experience in regard to the hospitals?

A.: Any case that is liable to take up extra time, or require extra visits, or is at all serious, or that may require detailed treatment, is sent into hospital.

Q.: What about the stitching of wounds?

A.: That is mainly done at the out-patients' department of the hospital.

Q.: And varicose veins and ulcers?

A.: If they are any size one would get them admitted to hospital.

Q.: Such work is done by the general practitioner here?

A.: Yes.

Q.: What about injections for varicose veins?

A.: The general practitioner would do it in Australia, but it is not done by the panel doctor in England.

Q.: Take the lancing of a boil?

A.: If it was a very small one a doctor may do it, but if it required an anæsthetic for opening, the case would be sent to a hospital.

Q.: You mean a local anæsthetic?

A.: No, a general anæsthetic. A simple boil you might put a knife into it.

Q.: Take medical cases like diabetes and pernicious anæmia?

A.: They would be sent to hospital for investigation and treatment.

Q.: What is the practice here?

A.: Here one would treat them oneself.

Q.: Take pneumonia in the early stages?

A.: In England, if there was any question of pneumonia, the case would be sent into hospital, whereas in Australia the doctor would keep it at home.

Q.: And chronic rheumatism?

A.: If the patient needed a lot of attention he would be sent to hospital; but I do not think you could get a hospital to take such a case in Australia.

Q.: Did you find it easy to get such cases into hospital in England?

A.: I never had any difficulty.

Q.: What has been your experience here?

A.: It is not difficult in regard to a hospital when I am on the staff, but it is not so easy in regard to other hospitals.

Q.: Could you get a case of chronic rheumatism admitted to a hospital here?

A.: I would not dream of trying, even if I were on the staff.

Q.: What about pneumonia?

A.: I could get such a case admitted if I were on the staff.

Q.: Did you have any influence at the hospitals in England?

A.: No; I was not on the staff of any.

Q.: What is the position there about panel doctors being on hospital staffs?

A.: Very few panel doctors are on the honorary staff at public hospitals.

Q.: How is the panel doctor regarded in England?

A.: The attitude of patients is that he is a panel doctor. It is meant as a somewhat derogatory term.

Q.: He is not of the social standing of the general practitioner in this country?

A.: No, definitely not.

The Chairman: I find it difficult to measure social standing. It is a fluctuating thing.

Mr. Abrahams: It cannot be measured in terms of pounds, shillings and pence perhaps. What about the professional status of panel doctors in England as compared with general practitioners here?

A.: The English panel doctor is not as professionally capable, not through any lack of intelligence or training in the first place, but through lack of doing the things which the general practitioner does here.

Dr. Mulvey: Does the panel doctor in England give many general anæsthetics?

A.: Except in dental cases he gives very few. Occasionally, if he gets a panel patient that can afford the fees at a private nursing home he may give an anæsthetic, but he gets no remuneration for that.

Q.: Give us an idea of how you carried on your practice during the busy part of the year at Plymouth, including your attendance of private patients?

A.: At Plymouth my consulting hours were from 9 to 10.30 in the morning and from 5.30 to 7.30 at night. At the busy time of the year I would see from 15 to 20 patients in the morning, while at night I might see anything from 30 to 60. That would include patients of all kinds.

Q.: And how many visits away from the surgery?

A.: The most I ever made away from the surgery in one day was 55. During the ordinary winter period the number would be between 30 and 40.

Q.: What did you find regarding the ailments of panel patients?

A.: As a rule they were of a minor kind, consisting of coughs, colds, mild influenza and rheumatism of the non-acute sort. That was very prevalent. There are also minor gastric disturbances.

Q.: What did your ordinary attendances consist of?

A.: You had to see the patient's record card to see if he was entitled to treatment. That is, until you got to know. Then you had to take out the clinical record card. You would have to enter the case, and whether it was an attendance or a visit. After you had seen the patient as quickly as possible you would write the clinical notes, issue a prescription, and a certificate if necessary.

Q.: What sort of an examination could you make if there were 60 patients to be attended to of an evening?

A.: I am afraid it was very cursory; in fact, I came to regard it merely as a job to ensure that I did not miss anyone who was seriously ill. Any such cases were usually packed off to the hospital.

Q.: Did you take temperatures?

A.: I generally put the thermometer in their mouths when they arrived and left it there while I was doing the clerical work. If it was a chest case I would apply the stethoscope. In one case when I told the patient to lift up his shirt so as to apply the stethoscope, he told me that the instrument could not be as good as Dr. So-and-So's, because with that instrument the doctor could hear through the patient's shirt.

Mr. Dovey: I suppose they have a sense of humour even in Plymouth?

A.: I think the remark was made seriously.

Dr. Sheldon was questioned by Mr. Dovey as to the standard of public hospitals in England.

Mr. Dovey: Are the public hospitals in Plymouth comparable with the public hospitals in the capital cities of Australia in regard to the standard of services given and the equipment?

A.: I should think that the standard was equally as good. It would depend a great deal on the particular consultants present in a particular town; it must vary according to that.

Q.: With the exception of the capital cities of Australia, the public hospitals in Plymouth would be superior?

A.: Yes, very definitely.

After further cross-examination by Mr. Dovey concerning panel practice in England and comparative conditions here and in England, during which Dr. Sheldon stated that almost as much time was taken up in his panel practice there in the keeping of records as in attendance on the patients, and that he had more time in Australia to devote to his patients than he had in England, he was further examined *in camera*.

Dr. Noel Rau, Brighton-le-Sands, stated that the bulk of his patients were working people. He was examined *in camera* and then was cross-examined by Mr. Dovey in open court. He said that he gave a greater number of services per patient to lodge patients than to private patients. "I think the lodge patients tend to come to the surgery for ailments which private patients would think twice about paying 10s. 6d. for, and I think they unnecessarily seek treatment." He found it worth his while to increase the numbers of his lodge patients, as it all went to the building up of the practice.

The Chairman: I do not think that this matter need be laboured. I am convinced that the general run of practitioners have regarded lodge practice as worth while in the circumstances. Partly for direct monetary return, and partly because it leads in the usual course to outside work for which they receive fees—I mean work outside the lodge services. I am going to assume, unless I am persuaded to the contrary, that practitioners have up to now, and would continue in present circumstances, to regard that sort of lodge practice as a desirable thing.

Mr. Dovey: You mention as one of the advantages what you call lodge "perks". You would expect in the same way to have perks coming from the patients on your list as insurance patients?

A.: Not to the same extent.

Q.: Why not?

A.: For instance, I notice that injection treatment is excluded. Quite a percentage of my income is for injection therapy. That is not payable under the panel system. Then there are minor operations, which form quite an appreciable part of the takings. They are excluded.

Q.: Is it your understanding that the scope of service to be provided by members of your profession under the national scheme is a more restricted service than the one under the friendly societies' contract, or is it the same, or is it wider?

A.: It is very much wider under the national scheme.

Q.: In what respect is it very much wider?

A.: The friendly society service excludes fractures, injection therapy and minor operations. There are also other chargeable things under the lodge scheme, such as certain kinds of certificates. Mileage does not affect me very much, but the scope there is fairly large.

Dr. Rau estimated the figure of 36·8% as the amount by which his private practice would diminish under national insurance. He was cross-examined by Mr. Dovey as to this figure and as to the method used in arriving at it. He was also examined by Mr. Williams as to the adequacy of lodge remuneration and as to his motives.

The Chairman: I confess that I am getting rather tired of this matter being brought up time after time. You know how we are going to find unless we have something quite unexpected brought before us. It does not matter what the considerations are which actuate medical men. They have thought it worth while, and will continue to think it worth while in similar circumstances, to do lodge business at something like the rate they have been receiving in the past. It really does not matter much whether they flatter themselves that they do it partly out of charity or partly by way of concession, or for any other reason. The whole point is, the British Medical Association suggests that there is going to be a very radical change in circumstances, which may justify them in saying that past rates, adequate though they were, will not be adequate in the future. That is the crux of the matter. Why this continual raking over of the adequacy in the past and the motives of the doctors, and what they choose to call it? This gentleman says it may be regarded partly as a charitable allowance. Another gentleman also used the word charitable and then thought it tactful to withdraw it and use the word "concessional". Will you be good enough, Dr. Rau, to withdraw the one and substitute the other?

Mr. Williams: I know that certain conditions will be different in the future. What I want to put to this witness is that, on the sole consideration of price, it was worth while.

The Chairman: It does not matter what he says. I find that they have considered it worth while from the point of view of business.

Mr. Williams: Your Honour will not find that that will be the case notwithstanding the changed conditions.

The Chairman: That is another matter. Can you not make this scratch? Let everyone assume for all purposes that on the average the present lodge rates are fair business rates, accepted by all as such. Then say that in the future, having regard to the changes of conditions which may take place under the insurance régime, what will be the fair thing.

Dr. Eric Alfred Tivey, Mosman, said that he had been in practice at Mosman for thirteen years and was medical officer to eight societies, with 195 members and 331 dependants. Questions as to examination of applicants for lodge membership, examination of their wives and as to income, as to the right fee and as to women taking more medicine than men, were by arrangement omitted. The witness was then examined *in camera* and the Commission adjourned at 4.30 p.m. until 10.30 a.m. on Monday, September 5, 1938.

Post-Graduate Work.

MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

THE Melbourne Permanent Post-Graduate Committee announces that courses of instruction in pathology, physiology and anatomy will be held during 1938. These courses have been arranged to suit the requirements of those intending to sit for the examinations for higher degrees and diplomas in August, 1939.

The course in anatomy will commence on Monday, September 19, 1938, and the courses in pathology and physiology on Monday, October 17, 1938. The courses will continue for the remainder of the year and for the first half of 1939.

The course in pathology is suitable for those desirous of obtaining the degree of Doctor of Medicine or Master of Surgery, or the Diploma in Gynæcology and Obstetrics, the Diploma in Therapeutic Radiology and Electrology, the Diploma in Diagnostic Electrology or the Diploma in Laryngology and Otology. The course in physiology is suitable for those studying for the degree of Master of Surgery or Doctor of Medicine, or the Diploma in Gynæcology and Obstetrics, Diploma in Diagnostic Radiology, Diploma in Public Health, Diploma in Ophthalmology or the Diploma in Psychiatric Medicine. The course in anatomy is suitable for study for the degree of Master of Surgery, the Diploma in Ophthalmology, Diploma in Laryngology and Otology, Diploma in Gynæcology and Obstetrics, Diploma in Therapeutic Radiology and Electrology, Diploma in Diagnostic Radiology and the Diploma in Psychiatric Medicine.

Those desirous of attending the courses should register and pay the necessary fees at the office of the Registrar, the University of Melbourne.

Correspondence.

NATIONAL HEALTH INSURANCE.

SIR: It is heartening to learn from your correspondence columns that there are many who advocate the complete rejection of national health insurance by the medical profession.

The Royal Commission has been set up primarily to determine what is a fair capitation fee. This, I submit, is merely a red herring drawn across the trail. The question of whether we get three shillings more or less is not important at all. Such a trifle will not matter to us when we have sacrificed our liberty for ever.

The technique of appointing a Royal Commission is a time-honoured one with politicians. It gets them out of a tight corner and helps them to shelve an awkward question. Then, when feelings have cooled down under the mellowing influence of time, we find the politicians quietly getting their own way without opposition.

The British Medical Association committee should not accept any Government proposal without a plebiscite of all British Medical Association members in Australia. In the meantime the subject should be kept actively before members so that we shall not be drugged into a state of acquiescence.

There is a strong body of opinion amongst general practitioners that the profession should reject the whole scheme. The pretended solicitude of the politicians for the health of the worker is in reality a solicitude for the health of the exchequer. The medical profession is to be thrown to the worker as a sop, in order to make the proposals more palatable. The medical benefit proposals are not essential to the working of the Act; they could be expunged completely without any inconvenience. An insured worker who requires sickness benefit could go to his private doctor for his certificates and his prescriptions for free medicine.

If the medical profession were to reject the proposals almost unanimously the Government would be forced to modify the scheme. No government would dare to force its proposals upon an unwilling body of men if they possessed greater voting power. Let us show by a plebiscite that we possess power enough to hinder the process of our own enslavement.

The profession here is not in the same state as was the profession in England before the institution of national insurance. There the Act raised the status and the income of a very large number of doctors. Here the Act would lower the income and the status of everybody.

Yours, etc.,

MICHAEL KELLY, M.B., B.S.

Bunbury,
Western Australia,
August 21, 1938.

SIR: I think that we should pause and take stock of ourselves at this juncture and realize that, individually, we have only ourselves to blame for the position we are in. It is very easy when things go wrong to criticize the people that have gone wrong, and I find that, casting my mind back to the pre-insurance days, very few took any interest in association of doctors (as such). We might grumble as we paid our fees; but our faults lie in our previous disinterestedness and lack of real union.

We have in the past, and still are, giving of ourselves so freely to the public that our very altruism has brought about our position in the present impasse, and astute politicians have taken note of it.

All the protests in the world will not make such an effect as the formation of a guild (or union if you like), including every member of the profession from his graduation, associated with but not part of the British Medical Association. Under full-time directors at a suitable salary, we could speak as one voice, and then governments would take notice, as they do of any other real organization.

One of the most capable of the medical minds of this State voiced this also as his opinion as the only hope the medical profession has of consolidating its position now and in the future, and however much we rebel, he is absolutely right.

Why do we stand aloof from such a plan? It is not incompatible with our dignity to sue for our just rights; why should it be beneath our dignity to ensure them in this manner? Two packets of cigarettes, or its equivalent, per week would bring this to fruition.

It can be done; it must be done! Let us start.

Yours, etc.,

J. L. SIMMONDS.

Crows Nest,
Queensland.
(Undated.)

SIR: After reading the précis of the proceedings of the Royal Commission on National Health Insurance published in this journal, August 20, 1938, I was disappointed to find a certain relevant statement by Mr. Green had been omitted. I refer to the table on page 305, analysing Mr. Green's calculation of the much-debated figure of 3·2. This table should have read as follows:

720	married males (between thirty and fifty years of age)	720
	Wives of married males	720
	Dependants of married males	1,440
280	unmarried males	280
1,000	members	3,160 persons.

The omitted statement is in parentheses. Mr. Green, in his remarks prior to giving this table, stated he had

taken this age-group as representative of friendly society membership, and that each of these families averaged two dependent children. Married males younger than thirty years or older than fifty years have a much lower dependent rate and were not considered. Their inclusion would lower the figure to 3·16. For this reason I think the omitted statement is of considerable interest. Without this knowledge those who know the census figures might be led to believe that Mr. Green had made an arithmetical error.

It is not my place to criticize evidence before the Commission, so I merely wish to draw attention to a relevant omission, which it is necessary to know, in order to understand how Mr. Green finally arrived at the figure of 3·2 instead of 2·5.

Yours, etc.,

M. O. KENT HUGHES.

36, Margaret Street,
Moonee Ponds,
August 23, 1938.

[The table on page 305 of the journal of August 20, 1938, was copied exactly from the transcript of Mr. Green's evidence.—EDITOR.]

SIR: In the letter written by Dr. Brettingham Moore, and published in your journal, is contained a frank statement of the position of the medical profession in relation to the Federal national health insurance scheme. If further evidence be necessary to emphasize the need for unity in our struggle against the imposition of bureaucratic control on our profession, it is contained in the report of its House of Delegates (May 11, 1938), published at the instance of the Medical Society of New York. The following quotation is taken from that report:

> It is the duty and prerogative of medical practitioners to provide competent professional service, properly controlled and disciplined by themselves, and not by laymen, nor by government bureaus or officials.
>
> It is equally the prerogative and duty of properly qualified non-medical persons or agencies to provide the facilities and financial support for this competent medical care with strict adherence to the three requirements specified in this report.
>
> These three requirements demanded of plans sponsored by laymen to finance medical service are:
>
> 1. That they maintain and raise the standard of quality of medical care.
>
> 2. That they provide a fair and reasonable remuneration on an ethical basis.
>
> 3. That they do not involve, directly or indirectly, the interposition of a third party, as regards medical matters, between the patient and the doctor of his choice.

The present Federal national health insurance scheme does not comply with any one of these essential requirements; hence we are forced unwillingly to oppose the Government by refusing to accept service. It cannot be stated too often that the medical profession will always be ready to cooperate with the Government in working a scheme of national health insurance, provided that the three essential requirements, referred to above, are not infringed in any particular.

Yours, etc.,

R. O. WILLIAMS.

194, Bourke Street,
Goulburn,
New South Wales.
August 27, 1938.

ANÆSTHESIA IN PULMONARY TUBERCULOSIS.

SIR: Activation of pulmonary tuberculosis after anæsthesia is caused, it has always seemed to me, by the deep breathing which so frequently occurs with some anæsthetics, and which would have the same effect as making a child with a tubercular knee run several miles.

Ether alone usually causes increased respiratory movements; but if oxygen is added, as Dr. Susman has said is done, in a closed circuit, the movements are much smaller.

The treatment of pulmonary tuberculosis is partly directed to resting the lungs as much as possible, so surely it is stupid to goad them with any anæsthetic which causes more than a minimal increase of pulmonary ventilation.

Yours, etc.,

T. MAYNARD FURBER.

143, Macquarie Street,
Sydney,
August 30, 1938.

SCHOPENHAUER AND HIS ILL HEALTH.

SIR: With reference to the statement made in *The British Medical Journal* of July 2, page 22, and Dr. F. D. Rolleston's letter in reply to same, I have seen it frequently stated that Schopenhauer had been infected when a student. He possibly did, but beyond getting a chancre never got another lesion. His pessimism was more likely due to another cause, the *Bacillus coli*, which apparently worried him all his life.

Two other notable literary men whose lives were possibly ruined by this infection were the Italian poet Leopardi and James Thomson, who translated Leopardi's works into English, as well as writing "The City of Dreadful Night".

Yours, etc.,

J. MORRIS ROE.

Victory Chambers,
Queen Street,
Brisbane.
August 24, 1938.

A WARNING.

SIR: I consider it my duty to inform the medical profession, through the columns of your journal, that a female impostor has recommenced a round of the Sydney public hospitals. I say recommenced, as I identified her as the subject of a letter by Dr. O. W. Mater published in your journal of August 15, 1936.

Last night a girl, about twenty-four years of age, presented herself in our casualty department suffering, apparently, from a profuse hæmatemesis. She vomited about two pints of blood before her admission to the ward. I saw her about one hour later in the ward, and then identified her as the impostor of two years ago. Her colour was good and her temperature and pulse were normal. She has a contracture of the left forearm and arm. An examination was then made of her belongings, and an eight-ounce bottle full of blood was found. This morning the blood was still fluid, and microscopically appeared to be mammalian. When confronted with the bottle *et cetera* she asked for her clothes and left.

I believe her former efforts at imposture were made with an aniline dye known as "Primrose Red", but her latest effort appears to be on a more elaborate scale, and includes mammalian blood, which has apparently been citrated. I think the underlying motive is to satisfy some form of addiction, probably morphine. I might add that she poses as a nurse, and has a uniform and badges *et cetera* in her suitcase.

Yours, etc.,

P. HUNT,
Medical Superintendent, Royal South Sydney Hospital.

Joynton Avenue,
Zetland,
New South Wales.
September 1, 1938.

THE ROYAL NORTH SHORE HOSPITAL OF SYDNEY.

SIR: The advertisement that appeared in THE MEDICAL JOURNAL OF AUSTRALIA of September 3, 1938, calling for applications to the whole of the positions on the honorary medical staff is a result of the repeal of that hospital's Act of incorporation and the reincorporation of the institution under the *Public Hospitals Act*, 1929-1937.

The board of the hospital has notified each member of the honorary medical staff that consequently his present position has become a temporary one and that he is free to apply for reappointment.

It is believed that all the active members of the staff will do so.

Yours, etc.,
F. GUY GRIFFITHS,
Chairman, Honorary Medical Staff, Royal North Shore Hospital of Sydney.

131, Macquarie Street,
Sydney,
September 5, 1938.

THE EMPIRE RHEUMATISM COUNCIL.

SIR: The Empire Rheumatism Council greatly appreciates your leading article of June 4 regarding its work, and trusts that as a result of your advocacy there will soon be an Australian army corps engaged in the war on rheumatism. Medical scientists in the home country, I can confidently state, have the highest respect for their colleagues in Australia, and anticipate from them valuable assistance in solving the problems of the causation of rheumatic disease and in determining the best means of treatment.

We have had within the past few months the advantage of visits from several Australian doctors (coming from Sydney, Melbourne and Adelaide), and all seemed to be satisfied that our plan of campaign is sound. An essential part of that plan, as Lord Horder stressed at our first annual meeting (December, 1937), is the formation of fully autonomous affiliated councils in the Dominions. There are two good reasons for this: firstly, the scientific reason that research, which must investigate methodically every possible factor of the problem, will have better prospects of success following coordinated efforts in areas presenting widely different conditions of climate and of environment; secondly, the "political" reason, that Australian energy is more likely to be effectively enlisted and employed under Australian direction.

May I venture to suggest that the first practical step would be to form in some of the principal States a small committee of medical scientists having a special interest in rheumatic disease; these State committees to keep in view the foundation at an early date of a central Federal body. The next step would be to seek the help of the lay public to provide a fighting fund and to contribute the aid of their administrative ability. In Great Britain we aim to make our general council about equally representative of the medical and of the lay community, giving to the latter a preponderance in matters of financial administration, reserving scientific questions to the former.

Our council would gladly assist in the formation of an Australian council in any way suggested. If I may be permitted a personal reference, as an Australian I appreciate greatly the honour of having been entrusted by the British medical fraternity with the organization of this Empire campaign against a group of diseases which inflicts on our race more suffering and more economic loss than any other; and I hope to see my native country taking its full part.

Yours, etc.,
FRANK FOX,
Organising Secretary,
Empire Rheumatism Council.

1, Mitre Court Buildings,
Temple,
London, E.C.4.
August 23, 1938.

Medical Societies.

REPATRIATION DEPARTMENT MEDICAL OFFICERS' ASSOCIATION.

THE annual meeting of the Repatriation Department Medical Officers' Association was held at Melbourne on August 5, 1938. The following officers were elected.

President: Dr. W. J. Beveridge.
Vice-President: Dr. N. Pryde.
Honorary Secretary: Dr. C. W. H. Farrow.
Honorary Treasurer: Dr. J. Smythe Yule.
Members of the Committee: Dr. G. O. Robertson, Dr. J. Deravin.
Trustees: Dr. K. Smith, Dr. A. H. Melville.
Auditors: Dr. S. McLennan, Dr. H. I. Gibb.

Among other business, national health insurance was discussed, and the following resolution was carried:

> That this Association is sympathetic to the British Medical Association's action in presentation of the case to the Royal Commission and urges support of individual members, each member to contribute according to his own inclination.

The number of members of the association has now reached 52.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Clipsham, Sidney Brookes, M.B., 1938 (Univ. Sydney), Brisbane General Hospital, Brisbane.
Franklands, Herbert Malcolm, M.B., B.S., 1934 (Univ. Melbourne), School of Public Health, Sydney.
Kenny, John Joseph Martin, M.B., B.S., 1934, M.D., 1937 (Univ. Melbourne), School of Public Health, Sydney.
McDonnell, John Thomas, L.L.M., 1916, R.C.P. (Ireland), L.L.M., 1916, R.C.S. (Ireland), c.o. A.M.A., 201, Macquarie Street, Sydney.
Moore, Reginald Lithgow, M.B., Ch.B., 1936 (Univ. New Zealand).
Birks, Walter Gordon, M.B., B.S., 1937 (Univ. Adelaide), Royal Alexandra Hospital for Children, Camperdown.
Panting, Rona May, M.B., B.S., 1934 (Univ. Melbourne), Vaucluse.
Forbes, Litton Arthur Armitage, M.B., B.S., 1922 (Univ. Melbourne), Woodburn.
McEwen, George, M.B., B.S., 1937 (Univ. Melbourne), Collarenebri.

NOTICES.

Renwick Hospital for Infants.

WE have been asked by the Secretary of the Benevolent Society of New South Wales to publish the following notice.

By a recent decision of the Board of Directors of the Benevolent Society of New South Wales, the age limit for indoor and outdoor patients of the Renwick Hospital for Infants, Summer Hill, has been increased from two to three years.

Lost Property.

The following information has been forwarded by the Medical Secretary of the Victorian Branch.

A bag containing instruments, drugs *et cetera* was found in Glen Street, Hawthorn, Victoria, on May 20, 1938, and is now in the possession of the Hawthorn police.

Diary for the Month.

SEPT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 20.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: Branch.
SEPT. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 23.—Queensland Branch, B.M.A.: Council.
SEPT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Victorian Branch, B.M.A.: Council.
SEPT. 29.—South Australian Branch, B.M.A.: Branch.
SEPT. 29.—New South Wales Branch, B.M.A.: Branch.
SEPT. 30.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates.
OCT. 4.—New South Wales Branch, B.M.A.: Council (Quarterly).
OCT. 5.—Western Australian Branch, B.M.A.: Council.
OCT. 5.—Victorian Branch, B.M.A.: Branch.
OCT. 6.—South Australian Branch, B.M.A.: Council.
OCT. 7.—Queensland Branch, B.M.A.: Branch.

Medical Appointments.

The following have been appointed Resident Medical Officers at the Adelaide Hospital, Adelaide: Dr. I. A. O. Fox, Dr. D. W. Brummitt, Dr. K. J. Basedow and Dr. H. A. Goode.

* * *

Dr. E. J. Ryan and Dr. H. N. Merrington have been appointed, pursuant to the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Act, of* 1933, of Queensland, members of the Medical Board constituted by that Act.

* * *

Dr. E. J. C. Claridge has been appointed a Certifying Medical Practitioner at Minyip, Victoria, in accordance with the provisions of the *Workers' Compensation Acts.*

* * *

Dr. H. L. Spearman has been appointed Honorary Out-Patient Psychiatrist to the Psychiatric Clinic, Broughton Hall, Department of Mental Hospitals of New South Wales.

* * *

Dr. C. P. O'Toole has been appointed Government Medical Officer at Bulahdelah, New South Wales.

* * *

Dr. F. J. Williams has been appointed a Member of the Executive Council for the Territory of Papua.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xx to xxii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Officers.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

DEPARTMENT OF PUBLIC HEALTH, WESTERN AUSTRALIA: Medical Officer.

THE BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, NEW SOUTH WALES: Acting Assistant Surgeon Superintendent.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.